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Academy for Emergency, Trauma & Critical Care Medicine

PALLIATIVE CARE & PAIN MANAGEMENT

AT YOUR FINGERTIPS



NMC CBME ALIGNED MD · DNB

CBME-Based MD Palliative Medicine Exam-Preparatory Manual

NMC competency-based curriculum · Pain management · 100 malignancies · Previous-year questions with answers · IAPC & Twycross essentials



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Pain & Palliative Medicine · Academic Quality, AIMS Kochi

EXPLORE BY SECTION — tap a box to jump

1 **Section 1** Complications in the Palliative Patient

2 **Section 2** Common Malignancies (100)

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Amrita School of Medicine · Amrita Institute of Medical Sciences, Kochi

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FOREWORD

Foreword

From Dr. Gireesh Kumar K. P. · To the postgraduates of Palliative Medicine



Dear postgraduate,

India adds roughly 1.3 million new cancer patients every year, and behind every one of them stands a second patient the textbooks rarely name – the family. Palliative Medicine is the specialty that treats both. It is not the medicine of "nothing more to be done"; it is the medicine of total pain, total symptom burden and total suffering – physical, psychological, social and spiritual – attended to with the same rigour a cardiologist brings to an ECG. The MD examination tests exactly that: can you titrate a syringe driver at 3 a.m., break bad news without breaking the person, and still write a scientifically defensible answer on the WHO analgesic ladder the next morning.

Palliative Care & Pain Management at Your Fingertips takes the National Medical Commission's competency-based curriculum for MD Palliative Medicine and maps it, topic by topic, to the way examiners actually ask – definitions first, then classification, then a management table, then a pearl worth the extra mark. Because pain is the single largest examinable domain in this specialty, and because the Department asked for it to stand apart from the general curriculum, **Pain Management occupies its own dedicated section** of this manual, with one full page devoted to each sub-topic, carrying its own drug table of *Drug · Indication · Dose · Caution* wherever pharmacotherapy is discussed.

The final part of the book is pure examination practice – rapid-fire viva questions, single-best-answer MCQs with explanations, solved clinical case scenarios, model long- and short-essay answers, and an algorithm atlas you should be able to redraw from memory under examination pressure.

My hope is simple: that this volume sits beside you through the long months of preparation, and that it helps you become the kind of palliative physician every patient facing a life-limiting illness deserves – one who listens, relieves suffering competently, and preserves dignity to the very end.

With best wishes for your preparation,

Dr. Gireesh Kumar K. P.
Principal, Amrita School of Medicine
Director, Academy for Emergency, Trauma & Critical Care Medicine (AETCM), Kochi
Kochi · 2027

HOW TO USE THIS MANUAL

- Read the previous-year-style questions first – they show how each topic is examined.
- Learn the definition word-perfect; examiners award the first marks for it.
- Reproduce one table and, where given, one flowchart in every long answer.
- Every topic that mentions a drug carries a standard *Drug · Indication · Dose · Caution* table – learn the table, not the paragraph.
- Close each pharmacology-heavy answer with a clinical pearl or a current guideline – that is where the extra marks are.

How to Use This Book

This manual follows the NMC Guidelines for Competency Based Postgraduate Training Programme for MD in Palliative Medicine. The theory examination has four papers – Paper 1: Basic Sciences as applied to Palliative Medicine; Paper 2: Principles and Practice of Palliative Medicine (Cancer); Paper 3: Principles and Practice of Palliative Medicine (Non-Cancer); Paper 4: Recent Advances in Palliative Medicine – followed by a clinical/ practical examination of one long case and three short cases, and an oral examination on drugs, instruments, radiological and clinical images, and charts.

Every Topic Page Contains	What It Covers
Previous-year-style questions	Examination-style questions in the pattern of MD/DNB Palliative Medicine theory and viva papers, built from the NMC syllabus weighting.
CBME competencies	What the postgraduate should be able to do – mapped to the NMC Cognitive (CD), Affective (AD) and Psychomotor (PD) domain codes.
Definition	A standard one- or two-line definition to open any answer.
Tables	Classification, clinical features, assessment tools, and – wherever a drug is mentioned – a standard Drug · Indication · Dose · Caution table.
Clinical pearls & exam focus	High-yield points, mnemonics and viva questions.
Guidelines & recent advances	Current WHO / IAPC / NMC reference points.

IMPORTANT NOTE

Doses given are for an average adult with normal hepatic and renal function unless stated otherwise, and must always be individually titrated and adjusted for age, renal/hepatic function, frailty, drug interactions and local institutional protocol. This manual is an examination-preparatory aid, not a prescribing reference; verify every dose against a current formulary (e.g. the Palliative Care Formulary, PCF) and institutional policy before clinical use.

HOW THIS EDITION IS ORGANISED

- **Sections 1–3** — the examinable text: complications, 100 common malignancies and a stand-alone Pain Management section.
- **Section 4** — algorithms, solved cases, model long- and short-essay answers, viva and MCQs. Every topic header carries a **Practice Qs ›** button, every question an **Answer ›** link, and every answer a **Read the text ›** link back.
- **Sections 5–6** — landmark trials and the official DNB papers, each question linked to the page that answers it.
- **Appendix** — the full NMC CBME curriculum; each syllabus item carries a page chip that jumps to the text. Competency codes (CD/AD/PD) printed in the text jump back here.
- The footer of every page carries **Index**, **‹ Cover** and **Contents ›** buttons.

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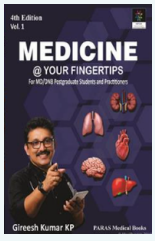
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SECTION 1

Infections, Electrolyte Disturbances & Other Complications in the Palliative Care Patient

Beyond pain and the malignancies covered elsewhere in this book, palliative care patients accumulate a predictable burden of infective, metabolic and other medical complications – from febrile neutropenia to hyponatraemia to venous thromboembolism – each requiring its own recognition and management approach, always weighed against the patient's overall goals of care.

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1.3 Other Medical Complications 64

AT A GLANCE

Section 1 at a glance — high-yield points

Problem	Must-know fact
Febrile neutropenia	Antibiotic within 1 hour; MASCC ≥ 21 = low risk
Hypercalcaemia	Saline then zoledronic acid 4 mg; PTHrP-mediated
Hyponatraemia / SIADH	Correct ≤ 10 mmol/L per 24 h; 3% saline for seizures
Tumour lysis	Rasburicase (not in G6PD deficiency), hydration
VTE / PE	LMWH or DOAC ≥ 6 months; stop in the dying
SVCO	Histology before steroids if lymphoma possible; stent
Delirium	Look for reversible causes — 30–50% reversible

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1.1.1 Febrile Neutropenia & Neutropenic Sepsis

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the emergency management of febrile neutropenia in a patient on chemotherapy. [Paper 1, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and urgently manage febrile neutropenia as an oncological emergency (CD4.1, PD3)

DEFINITION Febrile neutropenia is a single oral temperature ≥38.3°C (or ≥38.0°C sustained over 1 hour) with an absolute neutrophil count <500 cells/µL (or <1000 and falling), occurring 7-14 days after cytotoxic chemotherapy; it is a medical emergency with a mortality of 5-10% if treatment is delayed.

Risk Category	Approach
Low-risk (MASCC score ≥21, outpatient-eligible)	Oral broad-spectrum antibiotics (e.g. ciprofloxacin + amoxicillin-clavulanate) with close outpatient follow-up
High-risk	Admission for empirical IV broad-spectrum antibiotics within one hour of presentation, before culture results are available
Persistent fever at 4-7 days despite antibiotics	Add empirical antifungal cover and reimage/re-investigate for an occult source

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Piperacillin-Tazobactam	Empirical high-risk febrile neutropenia	4.5 g IV every 6-8 hours	Dose-adjust in renal impairment; broad empirical cover pending cultures
Cefepime	Alternative empirical monotherapy	2 g IV every 8 hours	Neurotoxicity (encephalopathy) in renal impairment if not dose-adjusted
Granulocyte Colony-Stimulating Factor (G-CSF)	Selected high-risk patients, or secondary prophylaxis for future cycles	5 µg/kg/day SC until count recovery	Bone pain is common; do not give concurrently with cytotoxic chemotherapy

★ CLINICAL PEARLS & EXAM FOCUS

► The first dose of empirical antibiotics should be given within one hour of a febrile neutropenic patient presenting – this single time target is one of the most frequently tested safety points in oncology.

► A normal chest X-ray or absent localising signs does NOT exclude serious infection in neutropenia – the inflammatory response needed to produce visible signs is itself blunted.

GUIDELINES & RECENT ADVANCES

- IDSA Clinical Practice Guideline for the Use of Antimicrobial Agents in Neutropenic Patients with Cancer (current version)

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1.1.2 Pneumonia in the Palliative Care Patient

Algorithm ›
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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the approach to pneumonia in a patient with advanced malignancy. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage respiratory infection in advanced illness, balancing treatment intensity with goals of care (CD5, PD3)

DEFINITION Pneumonia in palliative care patients may be community-acquired, aspiration-related (particularly in dysphagia, reduced consciousness or neurodegenerative disease), or healthcare-associated; management intensity should always be matched to the patient's overall prognosis and expressed wishes.

Clinical Context	Typical Approach
Reasonable prognosis, wishes active treatment	Standard empirical antibiotics per local pneumonia guidelines, with supportive oxygen/fluids as needed
Aspiration pneumonia, recurrent, poor swallow	Antibiotics covering oral anaerobes; speech-and-language swallow assessment; consider goals of care around feeding route
End-of-life/actively dying, symptom-focused goals	Oral/parenteral antibiotics may still relieve symptoms (fever, breathlessness) even without survival benefit – a reasonable palliative choice; comfort-focused care if antibiotics are declined or not wanted

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Amoxicillin-Clavulanate	Community-acquired/ aspiration pneumonia, oral option	625 mg orally 3 times daily, or 1.2 g IV 3 times daily	Dose-adjust in renal impairment; diarrhoea common
Ceftriaxone + Azithromycin	Hospitalised community-acquired pneumonia	Ceftriaxone 1-2 g IV once daily + azithromycin 500 mg once daily	QT prolongation with azithromycin, particularly in combination with other QT-prolonging drugs

★ CLINICAL PEARLS & EXAM FOCUS

► Even when cure is not the goal, a short course of antibiotics for pneumonia can be an effective symptom-control measure (reducing fever, cough, breathlessness) – the decision to treat is not simply binary.

► Recurrent aspiration pneumonia despite swallow precautions is itself a marker of advanced, often end-stage disease and should prompt a goals-of-care conversation.

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1.1.3 Urinary Tract Infection in Palliative Care

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the diagnosis and management of urinary tract infection in a palliative care patient with an indwelling catheter. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Distinguish asymptomatic bacteriuria from true infection and manage catheter-associated UTI (CD5, PD3)

DEFINITION Urinary tract infection is common in palliative care given frequent catheterisation, reduced mobility and immunosuppression; asymptomatic bacteriuria (a positive culture without symptoms) is extremely common with long-term catheters and should NOT be treated, a frequently missed distinction.

Presentation	Approach
Asymptomatic bacteriuria (catheterised, no symptoms)	No antibiotic treatment – treating positive urine cultures without symptoms promotes resistance without benefit
Symptomatic lower UTI (dysuria, frequency, suprapubic pain)	Short empirical antibiotic course per local susceptibility patterns
Catheter-associated febrile UTI/urosepsis	Catheter change plus systemic antibiotics; treat as sepsis if systemically unwell

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Nitrofurantoin	Uncomplicated lower UTI (avoid if CrCl <30 mL/min)	100 mg orally twice daily for 5 days	Pulmonary fibrosis with prolonged use, ineffective in renal impairment
Ciprofloxacin	Complicated UTI/urosepsis	500 mg orally twice daily, or 400 mg IV twice daily	Tendon rupture risk, QT prolongation, C. difficile risk

★ CLINICAL PEARLS & EXAM FOCUS

▸ A foul-smelling or cloudy catheter urine sample alone, without fever or systemic symptoms, is not an indication for antibiotics – this is one of the most over-treated 'infections' in palliative care.

EXAM-ORIENTED THEORY

Catheter-associated UTI — practical points

Issue	Recommendation
Diagnosis	Symptoms (fever, loin pain, delirium) + culture; dipstick unreliable with catheters
Specimen	Replace long-term catheter, then collect from the new catheter
Treatment	7 days if prompt response, 10–14 days if delayed; per local antibiogram
Recurrent UTI	Review catheter need, bladder washouts not recommended, consider suprapubic catheter
End of life	Treat for symptom relief; avoid investigations that do not change care

1.1.4 Catheter-Related Bloodstream Infection

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the recognition and management of catheter-related bloodstream infection in a patient with a central venous access device. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage central line-associated bloodstream infection (CD5, PD8)

DEFINITION Infection related to central venous access devices (ports, PICC lines, tunnelled catheters used for chemotherapy or long-term palliative infusions) presents with fever, rigors (classically with line flushing/ use), or exit-site inflammation, and requires a structured decision about whether the device can be salvaged or must be removed.

Organism / Severity	Approach
Coagulase-negative staphylococci, clinically stable	Antibiotic lock therapy plus systemic antibiotics may allow catheter salvage
Staphylococcus aureus, Candida species, or septic/unstable patient	Catheter removal is generally required alongside systemic antibiotics/ antifungals, given high complication rates if the device is retained
Tunnel/exit-site infection with purulence	Catheter removal; local wound care

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Vancomycin	Empirical cover for suspected Gram-positive line infection	15-20 mg/kg IV every 8-12 hours, levels monitored	Nephrotoxicity, red man syndrome if infused too rapidly
Fluconazole	Candida line infection (after removal)	400-800 mg IV/orally once daily	Hepatotoxicity, QT prolongation

★ CLINICAL PEARLS & EXAM FOCUS

▸ Candidaemia or Staphylococcus aureus bacteraemia from a line virtually always requires catheter removal – attempting to salvage the device in these organisms carries a high relapse/complication rate.

EXAM-ORIENTED THEORY

CRBSI — when to remove the line

Situation	Line management
S. aureus, Candida, Pseudomonas, mycobacteria	Remove the catheter
Tunnel / port-pocket infection, septic thrombophlebitis	Remove
Coagulase-negative staphylococci, uncomplicated	Can retain with antibiotic lock + systemic therapy
Persistent bacteraemia > 72 h	Remove; look for endocarditis
Prevention bundle	Chlorhexidine skin prep, aseptic access, daily review of need

1.1.5 Fungal Infections in the Immunocompromised Palliative Patient

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss invasive fungal infection risk and management in a neutropenic palliative care patient. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage invasive fungal infection risk in immunocompromised cancer patients (CD4.1, PD3)

DEFINITION Prolonged neutropenia, corticosteroid use and broad-spectrum antibiotic exposure predispose palliative care patients (particularly those with haematological malignancy) to invasive fungal infections – invasive aspergillosis, candidiasis, and in endemic/high-risk settings, mucormycosis.

Infection	Typical Clinical Clue
Invasive pulmonary aspergillosis	Persistent fever despite broad-spectrum antibiotics in prolonged neutropenia; nodular lesions with a 'halo sign' on CT chest
Invasive candidiasis	Persistent fungaemia, retinal lesions (endophthalmitis), hepatosplenic micro-abscesses
Mucormycosis	Rapidly progressive rhino-orbito-cerebral disease, black necrotic eschar; classically in uncontrolled diabetes/neutropenia

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Voriconazole	Invasive aspergillosis, first-line	6 mg/kg IV every 12 hours (day 1), then 4 mg/kg every 12 hours	Visual disturbance, hepatotoxicity, numerous drug interactions (CYP450)
Liposomal Amphotericin B	Mucormycosis, or voriconazole-refractory/ intolerant invasive fungal disease	5 mg/kg IV once daily (higher for mucormycosis)	Nephrotoxicity, infusion-related fever/rigors, hypokalaemia

★ CLINICAL PEARLS & EXAM FOCUS

▸ Persistent fever despite 4-7 days of appropriate broad-spectrum antibiotics in a neutropenic patient should prompt empirical antifungal therapy and imaging rather than simply escalating antibacterial cover further.

EXAM-ORIENTED THEORY

Antifungal agents at a glance

Drug	Spectrum / use	Key caution
Fluconazole	Candida (not krusei; reduced glabrata) — oral/oesophageal candidiasis	QTc, CYP3A4 interactions
Voriconazole	Aspergillus first line	Visual effects, hepatotoxicity, levels
Posaconazole	Prophylaxis in AML/MDS; mucor step-down	QTc
Echinocandins (caspofungin)	Candidaemia first line	Few interactions
Liposomal amphotericin B	Mucormycosis, cryptococcus, refractory mould	Nephrotoxicity, electrolyte loss

1.1.6 Oral Candidiasis & Mucosal Infections

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the management of oral candidiasis in a palliative care patient. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage common mucosal infections affecting oral intake and quality of life (CD3.1, PD3)

DEFINITION Oral candidiasis is extremely common in palliative care, predisposed by corticosteroid use (including inhaled), broad-spectrum antibiotics, xerostomia, dentures and immunosuppression; it causes pain, altered taste and reduced oral intake, directly affecting quality of life.

Presentation	Management
Pseudomembranous candidiasis (white plaques, wipe off)	Topical nystatin suspension or oral fluconazole for more extensive disease
Angular cheilitis	Topical antifungal/corticosteroid combination cream to the oral commissures
Oesophageal candidiasis (odynophagia, retrosternal discomfort)	Systemic (oral/IV) fluconazole given poor topical drug penetration to the oesophagus

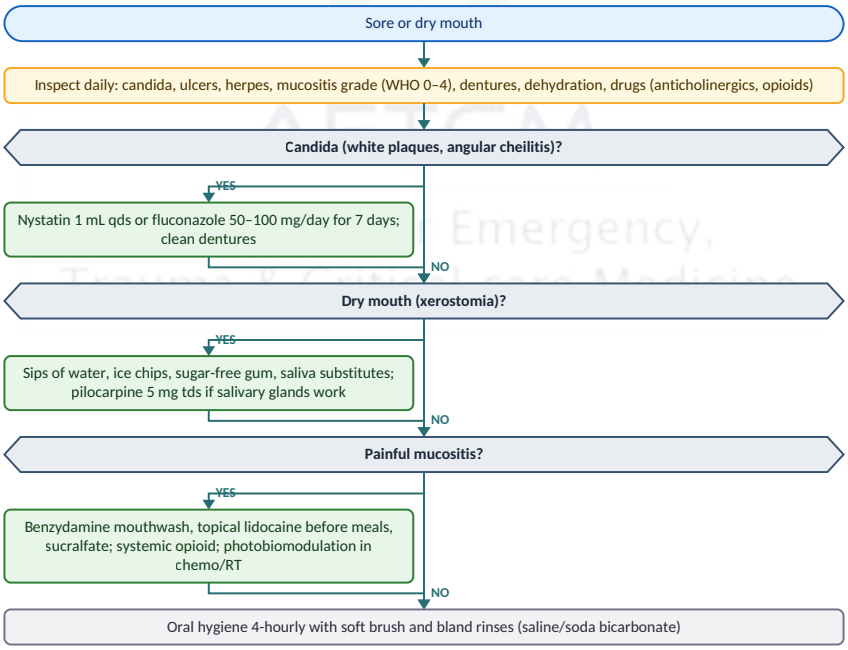
Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Nystatin Oral Suspension	Localised oral candidiasis	100,000 units (1 mL) 4 times daily, swish and swallow	Generally well tolerated; ensure adequate contact time with mucosa
Fluconazole	Extensive oral or oesophageal candidiasis	100-200 mg orally once daily for 7-14 days	Hepatotoxicity, QT prolongation, multiple drug interactions

★ CLINICAL PEARLS & EXAM FOCUS

► Rinse the mouth with water after using inhaled corticosteroids and brush/clean dentures regularly – simple preventive measures that substantially reduce recurrent oral candidiasis.

ALGORITHM · Dry Mouth and Oral Mucositis Algo 59 — full page ›



1.1.7 Skin & Soft Tissue Infections

Algorithm ›
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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the management of an infected pressure ulcer in a palliative care patient. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage skin and soft tissue infection including infected pressure ulcers and fungating wounds (CD6, PD8)

DEFINITION Cellulitis, infected pressure ulcers and secondarily infected fungating malignant wounds are common in palliative care given immobility, malnutrition, immunosuppression and disrupted skin integrity; distinguishing colonisation (expected in any open wound) from true infection requiring antibiotics is a key skill.

Presentation	Management
Cellulitis (spreading erythema, warmth, pain)	Systemic antibiotics covering Streptococcus and Staphylococcus species; mark the margin to monitor response
Colonised but non-infected chronic wound/ulcer	Wound care (debridement, dressings) alone – antibiotics are not indicated for colonisation without clinical infection
Malodorous fungating wound with anaerobic overgrowth	Topical or systemic metronidazole for odour control; charcoal dressings

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Flucloxacillin	Cellulitis, first-line	500 mg-1 g orally/ IV 4 times daily	GI upset; cross-reactivity in penicillin allergy
Metronidazole (Topical Gel)	Malodorous fungating wound, anaerobic odour control	Applied to wound bed once-twice daily	Local application generally well tolerated; systemic use requires avoiding alcohol

★ CLINICAL PEARLS & EXAM FOCUS

► Not every malodorous or discharging chronic wound is 'infected' in a way that needs systemic antibiotics – overtreating colonisation drives resistance without benefiting the patient; topical measures for odour/ exudate are often the more appropriate response.

EXAM-ORIENTED THEORY

Skin and soft-tissue infections in palliative care

Infection	Treatment
Cellulitis of lymphoedematous limb	Amoxicillin 500 mg tds (or clindamycin); 14 days; consider prophylaxis if ≥ 2 episodes/yr
Infected pressure ulcer	Debridement if appropriate; systemic antibiotics only for spreading cellulitis / sepsis
Fungating wound odour (anaerobes)	Topical / oral metronidazole
Necrotising fasciitis	Surgical emergency — discuss goals urgently
Scabies in debilitated patients	Permethrin 5% / ivermectin; treat contacts

1.1.8 Clostridioides difficile Infection

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the risk factors and management of Clostridioides difficile infection in an oncology patient. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and manage antibiotic-associated diarrhoea due to C. difficile (CD5, PD3)

DEFINITION C. difficile infection causes antibiotic-associated diarrhoea and colitis, with cancer patients at particular risk given frequent broad-spectrum antibiotic exposure, hospitalisation, and chemotherapy-related mucosal injury; severity ranges from mild diarrhoea to life-threatening fulminant colitis.

Severity	Management
Mild-moderate	Oral vancomycin or fidaxomicin; stop the inciting antibiotic if possible
Severe (marked leucocytosis, rising creatinine, hypoalbuminaemia)	Oral vancomycin at higher dose, with surgical consultation if deterioration
Fulminant (ileus, toxic megacolon, shock)	Oral/rectal vancomycin plus IV metronidazole; urgent surgical consultation for possible colectomy

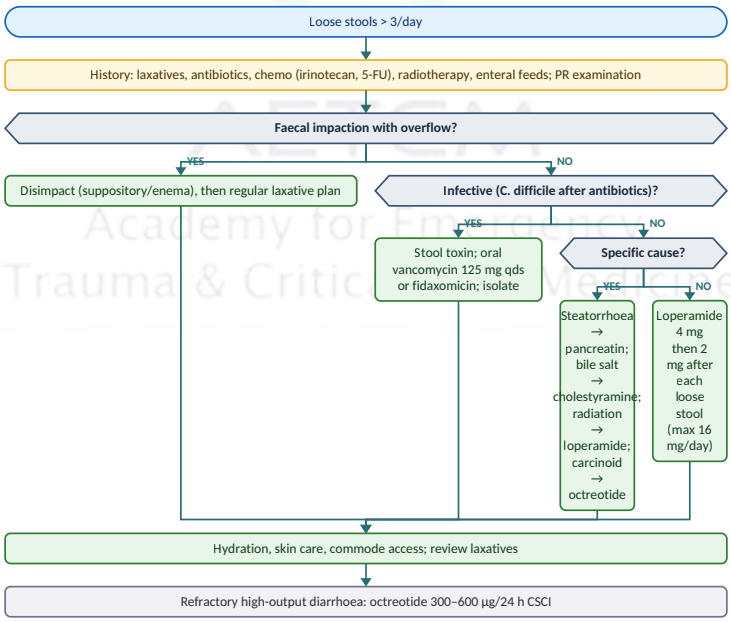
Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Oral Vancomycin	First-line, mild-severe disease	125 mg orally 4 times daily (higher for severe disease)	Minimal systemic absorption when given orally; poor efficacy if ileus prevents drug reaching the colon
Fidaxomicin	Alternative first-line, particularly for recurrence prevention	200 mg orally twice daily for 10 days	Expensive; lower recurrence rate than vancomycin in trials

★ CLINICAL PEARLS & EXAM FOCUS

► Avoid anti-motility agents (loperamide) in suspected C. difficile colitis, as they can precipitate toxic megacolon by slowing toxin clearance from the colon.

ALGORITHM · Diarrhoea in Palliative Care Algo 30 — full page ›



1.1.9 Herpes Zoster & Viral Reactivation in Cancer Patients

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the management of herpes zoster in an immunocompromised cancer patient. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage viral reactivation syndromes in immunosuppressed palliative care patients (CD4.1, PD3)

DEFINITION Cell-mediated immunosuppression from malignancy (particularly haematological), chemotherapy, corticosteroids and targeted/immunosuppressive agents (JAK inhibitors, high-dose steroids) predisposes to reactivation of latent viruses, most commonly varicella-zoster virus (shingles) and, less commonly, cytomegalovirus and hepatitis B.

Virus	Clinical Clue / Risk Context
Varicella-zoster virus (shingles)	Dermatomal vesicular rash with pain, sometimes preceding the rash; risk of disseminated disease in significant immunosuppression
Hepatitis B	Reactivation risk with rituximab and other B-cell-depleting therapy; screening mandatory before treatment
Cytomegalovirus	Reactivation particularly after allogeneic stem cell transplantation or intensive immunosuppression

Drugs used — Drug · Indication · Dose · Caution

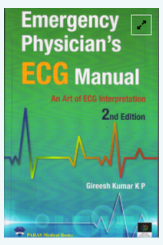
Drug	Indication	Dose	Caution
Aciclovir	Herpes zoster, standard dose	800 mg orally 5 times daily for 7-10 days (or IV for disseminated/severe disease)	Dose-adjust in renal impairment; nephrotoxicity with rapid IV infusion if inadequately hydrated
Valganciclovir	CMV reactivation/ disease	900 mg orally twice daily (induction)	Myelosuppression, particularly problematic in already cytopenic patients

★ CLINICAL PEARLS & EXAM FOCUS

► Start antiviral therapy for shingles as early as possible (ideally within 72 hours of rash onset) to reduce the risk of disseminated disease and post-herpetic neuralgia, particularly important in immunosuppressed patients.

► Hepatitis B screening before rituximab (or other B-cell-depleting therapy) is mandatory, as covered in Section 4 – reactivation can be fatal if missed.

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1.1.10 Antimicrobial Stewardship & End-of-Life Antibiotic Decisions

Algorithm ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the ethical and clinical considerations in deciding whether to start antibiotics in a dying patient. [Paper 3, ethics]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply antimicrobial stewardship principles and goals-of-care reasoning to infection management at the end of life (AD2, PD2, PD3)

DEFINITION Antimicrobial stewardship means using antibiotics only when they are likely to benefit the patient, at the narrowest effective spectrum and shortest effective duration; in the actively dying patient, the decision to start, continue or stop antibiotics should be driven by symptom benefit and the patient's goals of care, not reflexively.

Scenario	Reasoning
Reversible infection, reasonable prognosis	Treat with standard antibiotics aiming for cure/control, as in any patient
Actively dying, infection causing distressing symptoms (fever, breathlessness)	A trial of antibiotics may still be reasonable purely for symptom relief, distinct from a survival-prolongation goal
Actively dying, infection is one of several concurrent terminal processes	Antibiotics may add burden (IV access, monitoring) without meaningful symptom benefit – comfort-focused management alone may be more appropriate

A PRACTICAL FRAMEWORK

- Ask: is there a realistic chance this treatment will achieve the patient's goals (cure, prolonged meaningful time, or symptom relief)?
- Discuss explicitly with the patient/family what starting or not starting antibiotics is expected to achieve, avoiding the false framing of 'doing everything' versus 'doing nothing'.
- A time-limited trial of antibiotics with a pre-agreed review point can help when prognosis or goals are unclear.

★ CLINICAL PEARLS & EXAM FOCUS

► Decisions about antibiotics at the end of life are not simply 'treat' or 'don't treat' – they should be framed around whether the specific expected benefit (symptom relief vs cure vs prolonged survival) matches the patient's own goals.

► Stopping antibiotics that are not achieving the patient's goals is an active, considered clinical decision, not abandonment of care.

GUIDELINES & RECENT ADVANCES

- WHO Global Action Plan on Antimicrobial Resistance; institutional antimicrobial stewardship policy

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1.1.11 Sepsis and Septic Shock in the Cancer Patient

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** Outline the recognition and initial management of sepsis in a patient with advanced cancer. [Paper 2]
- **Q** How do goals of care modify sepsis management near the end of life? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Recognise sepsis early and deliver the hour-1 bundle when appropriate (CD4.1, PD3)
- ✓ Integrate ceilings of care and family communication into acute infection management (CD9, AD6)

DEFINITION Sepsis is life-threatening organ dysfunction caused by a dysregulated host response to infection (SOFA rise ≥ 2); septic shock is sepsis needing vasopressors to keep MAP ≥ 65 mmHg with lactate > 2 mmol/L despite adequate fluids (Sepsis-3).

Hour-1 bundle (Surviving Sepsis Campaign)

Step	Action
Measure lactate	Re-measure if > 2 mmol/L
Blood cultures	Before antibiotics (do not delay therapy)
Broad-spectrum antibiotics	Within 1 h of recognition
Fluids	30 mL/kg crystalloid for hypotension or lactate ≥ 4
Vasopressors	Noradrenaline if MAP < 65 mmHg after fluids

Goals-of-care framework

Situation	Approach
Good performance status, active treatment	Full sepsis bundle, ICU if needed
Advanced illness, uncertain trajectory	Time-limited trial of antibiotics/fluids; agreed ceiling (ward-based)
Actively dying	Comfort care; antipyretic, analgesia; antibiotics only if they relieve symptoms

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Piperacillin–tazobactam	Empirical, neutropenic or abdominal source	4.5 g IV 6–8-hourly	Renal dose adjustment
Meropenem	Suspected ESBL / severe sepsis	1 g IV 8-hourly	Seizure threshold; reserve per stewardship
Noradrenaline	Septic shock	0.05–0.5 µg/kg/min IV (central line ideally)	Extravasation; ICU setting

★ CLINICAL PEARLS & EXAM FOCUS

- ► qSOFA (RR ≥ 22, altered mentation, SBP ≤ 100) flags risk outside the ICU but is not diagnostic.
- ► A documented time-limited trial avoids both over-treatment and abandonment.

GUIDELINES & RECENT ADVANCES

- Surviving Sepsis Campaign Guidelines 2021
- ICMR Treatment Guidelines for Antimicrobial Use in Common Syndromes (2022)

1.1.12 Pneumocystis jirovecii Pneumonia (PJP)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Pneumocystis pneumonia in cancer patients — risk factors, diagnosis and prophylaxis. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Identify patients needing PJP prophylaxis and treat PJP (CD4.1, CD2)

DEFINITION PJP is an opportunistic fungal pneumonia in patients with impaired cell-mediated immunity — prolonged steroids (≥ 20 mg prednisolone for ≥ 4 weeks), haematological malignancy, fludarabine, temozolomide with radiotherapy, stem-cell transplant.

Clinical features and diagnosis

Feature	Detail
Presentation	Subacute dry cough, breathlessness, fever; hypoxia out of proportion to signs; desaturation on exertion
Imaging	Bilateral perihilar ground-glass opacities on CT; cysts; pneumothorax risk
Diagnosis	Induced sputum / BAL immunofluorescence or PCR; raised β-D-glucan, LDH

Prophylaxis indications

Group	Prophylaxis
Prednisolone ≥ 20 mg/day for ≥ 4 weeks	Co-trimoxazole 960 mg three times weekly
ALL, allogeneic HSCT, fludarabine, alemtuzumab	Co-trimoxazole; dapsone or atovaquone if intolerant
Temozolomide + radiotherapy	Co-trimoxazole during treatment

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Co-trimoxazole	Treatment	15–20 mg/kg/day trimethoprim component IV/ oral in 3–4 doses × 21 days	Hyperkalaemia, marrow suppression, rash
Prednisolone	PaO2 < 70 mmHg	40 mg bd × 5 d, 40 mg od × 5 d, 20 mg od × 11 d	Hyperglycaemia
Primaquine + clindamycin	Co-trimoxazole intolerance	Primaquine 30 mg od + clindamycin 600 mg 8-hourly	Check G6PD

★ CLINICAL PEARLS & EXAM FOCUS

- ► Non-HIV PJP has a higher mortality (30–50%) and more abrupt onset than HIV-associated PJP.
- ► Dexamethasone used for brain metastases is an under-recognised PJP risk.

GUIDELINES & RECENT ADVANCES

- ECIL-5 guidelines on PJP in haematology patients
- IDSA/ASCO outpatient febrile neutropenia & prophylaxis guidance

1.1.13 Invasive Aspergillosis and Mucormycosis

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss invasive mould infections in neutropenic patients, including mucormycosis. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Suspect and initiate treatment for invasive fungal infection in high-risk patients (CD4.1)

DEFINITION Invasive mould infections occur in prolonged neutropenia (> 10 days), haematological malignancy, transplant and high-dose steroids; mucormycosis is additionally associated with uncontrolled diabetes and iron overload — a major issue in India.

Comparison

Feature	Aspergillosis	Mucormycosis
Typical site	Lungs, sinuses	Rhino-orbito-cerebral, lungs
CT clue	Nodules with halo sign; air-crescent later	Reverse halo sign; bony erosion in sinuses
Biomarker	Serum/BAL galactomannan positive	Galactomannan negative; tissue diagnosis (broad aseptate hyphae)
First-line drug	Voriconazole or isavuconazole	Liposomal amphotericin B + urgent surgical debridement

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Voriconazole	Invasive aspergillosis	6 mg/kg IV 12-hourly × 2, then 4 mg/kg 12-hourly; oral 200 mg bd	Visual disturbance, hepatotoxicity; many CYP interactions — monitor levels
Liposomal amphotericin B	Mucormycosis	5–10 mg/kg/day IV	Nephrotoxicity, hypokalaemia, hypomagnesaemia
Posaconazole	Prophylaxis (AML/MDS) or step-down	300 mg/day (delayed-release tablet)	QTc, interactions

★ CLINICAL PEARLS & EXAM FOCUS

► Black necrotic eschar on the palate or nasal turbinate in a diabetic or neutropenic patient = mucormycosis until proven otherwise.

► In advanced cancer, discuss the burden of disfiguring surgery honestly.

GUIDELINES & RECENT ADVANCES

- ECIL-6 / ESCMID–ECMM guidelines for aspergillosis and mucormycosis
- ICMR advisory on mucormycosis (2021)

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1.1.14 Tuberculosis in the Patient with Cancer

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss tuberculosis in patients with malignancy in India. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Diagnose and treat TB in immunocompromised palliative patients, including drug interactions (CD4.2, CD2)

DEFINITION Malignancy, chemotherapy, steroids and anti-TNF therapy increase the risk of reactivation of latent TB; in India TB frequently mimics or coexists with cancer (lung masses, lymphadenopathy, effusions).

Diagnostic approach

Situation	Test
Pulmonary	Sputum CBNAAT / Truenat (Xpert MTB/RIF) + culture; CXR / CT
Lymph node / effusion	FNAC or biopsy for histology + CBNAAT; pleural ADA > 40 U/L suggests TB
Latent TB before immunosuppression	IGRA or TST; consider preventive therapy per NTEP

Key drug interactions of rifampicin

Drug	Effect / action
Methadone, fentanyl, oxycodone	Levels fall — opioid withdrawal / pain crisis; increase dose
Dexamethasone	Halve effect — double steroid dose
Tyrosine kinase inhibitors, warfarin, azoles	Reduced levels — check interactions

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
HRZE (fixed-dose combination)	Drug-sensitive TB, intensive phase	Weight-banded FDC daily × 2 months	Hepatotoxicity; monitor LFTs, vision (ethambutol)
HRE continuation	Continuation phase	Weight-banded FDC × 4 months	Rifampicin interactions
Pyridoxine	Prevent isoniazid neuropathy	10–25 mg/day	—

★ CLINICAL PEARLS & EXAM FOCUS

- ► Never label a lung mass 'cancer' in India without considering TB — and vice versa.
- ► Rifampicin is the most important interacting drug in palliative prescribing.

GUIDELINES & RECENT ADVANCES

- National TB Elimination Programme (NTEP) Technical and Operational Guidelines
- WHO consolidated guidelines on TB (Module 4: treatment)

1.1.15 Hepatitis B Reactivation during Cancer Therapy

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Hepatitis B reactivation — at-risk patients, screening and prophylaxis. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Screen and protect patients receiving immunosuppressive therapy (CD4.1)

DEFINITION HBV reactivation is an abrupt rise in HBV DNA (± hepatitis flare) in HBsAg-positive or HBsAg-negative/anti-HBc-positive patients receiving chemotherapy, rituximab, steroids or stem-cell transplant; can cause fulminant hepatitis.

Risk stratification

Risk	Example	Action
High (> 10%)	Rituximab / anti-CD20, HSCT, HBsAg+ on any chemotherapy	Prophylactic entecavir / tenofovir
Moderate (1–10%)	HBsAg−/anti-HBc+ on TKIs or moderate steroids	Prophylaxis or HBV DNA monitoring
Low (< 1%)	Low-dose steroids < 1 week	Monitor

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Entecavir	Prophylaxis/ treatment	0.5 mg orally daily; continue 12–18 months after therapy ends	Dose-adjust in renal impairment
Tenofovir alafenamide	Alternative	25 mg daily	Renal/bone safer than TDF

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Screen HBsAg and anti-HBc in every patient before chemotherapy or rituximab.
- ▸ Lamivudine is no longer preferred because of resistance.

GUIDELINES & RECENT ADVANCES

- ASCO Provisional Clinical Opinion on HBV screening (2020)
- INASL guidance on HBV in immunosuppressed patients

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1.1.16 Cytomegalovirus and Herpes Simplex Infections

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Viral infections in immunocompromised palliative patients — CMV and HSV. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and treat common viral infections in immunosuppressed patients (CD4.1)

DEFINITION Herpes viruses reactivate with impaired T-cell immunity: HSV causes painful oral/genital ulcers and oesophagitis; CMV causes viraemia, pneumonitis, colitis and retinitis, chiefly after HSCT, alemtuzumab or high-dose steroids.

Presentations

Virus	Presentation	Diagnosis
HSV	Painful oral ulcers (often mistaken for mucositis), oesophagitis, genital ulcers	Swab PCR
VZV	Dermatomal or disseminated zoster	Clinical ± PCR
CMV	Fever, cytopenias, pneumonitis, colitis (diarrhoea, bleeding), retinitis	Quantitative CMV PCR; biopsy (owl's-eye inclusions)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Aciclovir	HSV mucocutaneous	400 mg oral 5×/day × 7–10 d (IV 5 mg/kg 8-hourly if severe)	Hydrate; renal dose
Valaciclovir	HSV/VZV, prophylaxis	500 mg bd (prophylaxis) / 1 g tds (zoster)	Renal adjustment
Ganciclovir	CMV disease	5 mg/kg IV 12-hourly × 2–3 weeks	Myelosuppression
Valganciclovir	CMV pre-emptive / maintenance	900 mg bd then od	Myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Treat oral 'mucositis' that fails to settle with antiviral cover — HSV is common.
- ▸ Letermovir is used for CMV prophylaxis after allogeneic HSCT.

GUIDELINES & RECENT ADVANCES

- ECIL-7 guidelines on herpesviruses
- NCCN Prevention and Treatment of Cancer-Related Infections

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1.1.17 Neutropenic Enterocolitis (Typhlitis)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Neutropenic enterocolitis — clinical features and management. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise abdominal emergencies in neutropenic patients (CD4.1, PD3)

DEFINITION Neutropenic enterocolitis is necrotising inflammation of the caecum and adjacent bowel during profound neutropenia (often after cytarabine or taxanes), presenting with fever, right iliac fossa pain and diarrhoea.

Assessment

Item	Detail
Clinical	Fever, RIF pain/tenderness, distension, diarrhoea ± blood, ANC < 500
Imaging	CT: bowel-wall thickening > 4 mm (caecum), pneumatosis, perforation
Differentials	C. difficile, CMV colitis, appendicitis, ischaemic colitis

Management

Step	Detail
Supportive	Bowel rest, IV fluids, NG decompression if ileus, transfusion
Antimicrobials	Cover gram-negatives and anaerobes (piperacillin–tazobactam or meropenem) ± antifungal
Surgery	Perforation, uncontrolled bleeding, clinical deterioration
Avoid	Anticholinergics, opioid excess and laxatives that worsen ileus

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Piperacillin–tazobactam	Empirical cover	4.5 g IV 6–8-hourly	Renal adjustment
Metronidazole	Added anaerobic cover (with cefepime)	500 mg IV 8-hourly	Peripheral neuropathy with prolonged use
G-CSF	Selected patients	5 µg/kg/day SC	Bone pain

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Bowel wall > 10 mm on CT predicts higher mortality.
- ▸ Avoid colonoscopy and enemas — perforation risk.

GUIDELINES & RECENT ADVANCES

- MASCC/ESMO guidance on gastrointestinal complications of neutropenia

1.1.18 Acute Cholangitis and Biliary Stent Infection

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Management of obstructive jaundice and cholangitis in pancreaticobiliary cancer. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage biliary obstruction and infection in palliative patients (CD4.1, CD3)

DEFINITION Acute cholangitis is infection of an obstructed biliary tree (tumour, blocked stent) presenting with Charcot's triad — fever, jaundice, right upper quadrant pain — and, when severe, Reynolds' pentad (shock, confusion).

Tokyo Guidelines 2018 severity

Grade	Criteria	Management
I (mild)	Responds to initial therapy	Antibiotics; elective drainage
II (moderate)	WBC > 12 000, fever ≥ 39 °C, age ≥ 75, bilirubin ≥ 5 mg/dL, albumin low	Early drainage (ERCP / PTBD) within 24–48 h
III (severe)	Organ dysfunction (shock, confusion, renal, coagulopathy)	Resuscitation + urgent drainage

Palliative considerations

Issue	Approach
Blocked plastic stent	Exchange for metal stent if prognosis > 3 months
Pruritus from jaundice	Drainage is the best treatment; see pruritus algorithm
End of life	Antibiotics for symptom relief; avoid invasive drainage if dying

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Piperacillin–tazobactam	Moderate–severe cholangitis	4.5 g IV 6–8-hourly	Renal adjustment
Ceftriaxone + metronidazole	Mild–moderate	2 g IV daily + 500 mg 8-hourly	Biliary sludge with ceftriaxone
Ciprofloxacin	Oral step-down / recurrent prophylaxis	500 mg bd	Tendinopathy, QTc

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Biliary drainage, not antibiotics alone, is definitive treatment.
- ▸ Recurrent cholangitis signals stent occlusion — usually after 3 months for plastic stents.

GUIDELINES & RECENT ADVANCES

- Tokyo Guidelines 2018 for acute cholangitis
- ESGE guideline on biliary stenting in malignant obstruction

1.1.19 Empyema and Infected Malignant Pleural Effusion

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss pleural infection in patients with indwelling pleural catheters. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage pleural infection in cancer patients (CD4.1, PD8)

DEFINITION Pleural infection is bacterial invasion of the pleural space — frank pus (empyema) or fluid with pH < 7.2, glucose < 3.3 mmol/L or positive culture; risk is higher with indwelling pleural catheters and after repeated aspiration.

Light's and pleural infection criteria

Test	Finding
Exudate	Protein ratio > 0.5, LDH ratio > 0.6, LDH > 2/3 upper limit
Complicated parapneumonic / empyema	pH < 7.2, glucose < 3.3, LDH > 1000, pus, positive Gram stain
RAPID score	Renal (urea), Age, Purulence, Infection source, Dietary (albumin) — predicts mortality

Management

Step	Detail
Drainage	Small-bore chest drain; IPC can be left in place and drained daily
Fibrinolysis	tPA 10 mg + DNase 5 mg intrapleural bd × 3 days (MIST-2)
Surgery	VATS decortication if fit and failing medical therapy

Drugs used — Drug · Indication · Dose · Caution

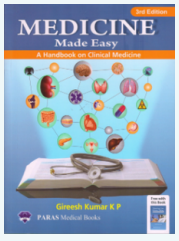
Drug	Indication	Dose	Caution
Co-amoxiclav	Community-acquired pleural infection	1.2 g IV 8-hourly → 625 mg tds oral	Total 3–6 weeks
Piperacillin–tazobactam	Hospital-acquired	4.5 g IV 8-hourly	Renal adjustment
Metronidazole	Anaerobic cover with cephalosporin	400 mg tds oral	Alcohol interaction

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Aminoglycosides penetrate pleura poorly — avoid.
- ▸ IPC-related infection rarely needs catheter removal.

GUIDELINES & RECENT ADVANCES

- BTS Guideline for Pleural Disease 2023



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1.1.20 Bacterial Meningitis and CNS Infection in Cancer

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Meningitis in immunocompromised patients — organisms and empirical therapy. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Initiate empirical therapy for CNS infection in high-risk patients (CD4.1)

DEFINITION CNS infection in cancer patients includes bacterial meningitis (Listeria in the immunosuppressed), post-neurosurgical / shunt infections, cryptococcal meningitis and brain abscess; carcinomatous meningitis is an important mimic.

Organisms by risk

Risk group	Likely organisms
Steroids, lymphoma, elderly	S. pneumoniae, N. meningitidis, Listeria monocytogenes
Post-neurosurgery / Ommaya reservoir	Staphylococci, gram-negative bacilli
T-cell defect	Cryptococcus, Toxoplasma, Nocardia

CSF patterns

Condition	CSF
Bacterial	Neutrophils, low glucose, high protein
Cryptococcal	Lymphocytes; India ink / CrAg positive; high opening pressure
Leptomeningeal carcinomatosis	Malignant cells on cytology (repeat ×3), high protein

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Ceftriaxone	Empirical	2 g IV 12-hourly	—
Ampicillin	Add for Listeria cover	2 g IV 4-hourly	Penicillin allergy → co-trimoxazole
Vancomycin	Penicillin-resistant pneumococcus / post-surgical	15–20 mg/kg IV 8–12-hourly	Levels, nephrotoxicity
Dexamethasone	Pneumococcal meningitis	10 mg IV 6-hourly × 4 d (with first antibiotic)	Not in cryptococcal meningitis

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Do not delay antibiotics for CT or lumbar puncture.
- ▸ Cryptococcal meningitis: amphotericin + flucytosine induction, then fluconazole.

GUIDELINES & RECENT ADVANCES

- ESCMID guideline on acute bacterial meningitis
- IDSA cryptococcal disease guidelines

1.1.21 Dengue, Malaria and Tropical Fevers in Cancer Patients

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Approach to fever in a cancer patient in India — tropical infections. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Consider regional infections in febrile immunocompromised patients (CD4.1, CD9)

DEFINITION In India, dengue, malaria, scrub typhus, leptospirosis and enteric fever cause fever in cancer patients and can mimic neutropenic sepsis or disease progression; thrombocytopenia from dengue compounds chemotherapy-related cytopenias.

Tropical fevers

Infection	Clue	Test	Treatment
Dengue	Myalgia, rash, falling platelets, rising haematocrit	NS1 (day 1–5), IgM	Fluids, monitor; avoid NSAIDs
Malaria	Cyclical fever, anaemia, travel/endemic area	Smear, rapid antigen	Artesunate IV (severe); ACT
Scrub typhus	Eschar, lymphadenopathy, pneumonitis	IgM ELISA	Doxycycline 100 mg bd
Leptospirosis	Flood exposure, jaundice, AKI	IgM ELISA	Penicillin / ceftriaxone / doxycycline
Enteric fever	Step-ladder fever, relative bradycardia	Blood culture	Ceftriaxone / azithromycin

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Artesunate	Severe malaria	2.4 mg/kg IV at 0, 12, 24 h, then daily	Delayed haemolysis
Doxycycline	Scrub typhus / leptospirosis	100 mg bd × 7 days	Oesophagitis; take upright
Azithromycin	Enteric fever / scrub typhus alternative	500 mg–1 g daily	QTc

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Dengue platelet counts may be misattributed to chemotherapy — check NS1 in the monsoon season.
- ▸ Paracetamol, not NSAIDs, for fever when dengue is possible.

GUIDELINES & RECENT ADVANCES

- National Centre for Vector Borne Diseases Control guidelines
- ICMR Standard Treatment Workflows — acute febrile illness

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1.1.22 Multidrug-Resistant Organisms and Infection Control

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q MDR organisms in palliative care — infection control and stewardship. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply infection-control and stewardship principles in palliative units (CD9, AD10)

DEFINITION Multidrug-resistant organisms (ESBL and carbapenem-resistant Enterobacterales, MRSA, VRE, Acinetobacter) are highly prevalent in Indian hospitals; repeated admissions, catheters and antibiotics put palliative patients at risk.

Common MDR organisms

Organism	Treatment options	Control
ESBL Enterobacterales	Carbapenem; piperacillin–tazobactam (some)	Contact precautions
CRE (KPC, NDM, OXA-48)	Ceftazidime–avibactam ± aztreonam (NDM), colistin, tigecycline	Isolation, screening
MRSA	Vancomycin, linezolid, teicoplanin	Decolonisation (mupirocin, chlorhexidine)
Acinetobacter (CRAB)	Colistin, sulbactam-based regimens	Strict hand hygiene

Palliative stewardship

Principle	Practice
Treat symptoms, not swabs	Colonisation needs no antibiotic
Time-limited trials	Review antibiotics at 48–72 h
Dignity	Isolation should not mean abandonment at end of life

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Colistin	CRE / CRAB	9 MU loading, then 4.5 MU IV 12-hourly	Nephro- and neurotoxicity
Linezolid	MRSA / VRE	600 mg bd oral/IV	Thrombocytopenia; serotonin syndrome with SSRIs

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Hand hygiene is the single most effective infection-control measure.
- ▸ End-of-life antibiotic use is high and often non-beneficial — ask 'whose goal does this serve?'

GUIDELINES & RECENT ADVANCES

- ICMR Antimicrobial Stewardship Programme guidelines
- National Action Plan on AMR (India)

1.1.23 Herpes Zoster and Post-herpetic Neuralgia (Pain Focus)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Post-herpetic neuralgia — prevention and management in cancer patients. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage acute zoster pain and prevent post-herpetic neuralgia (CD3.1, CD4.1)

DEFINITION Post-herpetic neuralgia is dermatomal pain persisting > 90 days after the zoster rash; risk rises with age, immunosuppression and severe acute pain.

Pain management of zoster

Phase	Management
Acute zoster	Antiviral within 72 h; paracetamol/NSAID; opioid if severe; consider early gabapentinoid
PHN — first line	Pregabalin / gabapentin, amitriptyline / nortriptyline, lidocaine 5% plaster
PHN — refractory	Capsaicin 8% patch, opioids (tramadol/tapentadol), nerve blocks
Prevention	Recombinant zoster vaccine (non-live) in at-risk adults

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Lidocaine 5% plaster	Localised PHN with allodynia	Up to 3 plasters 12 h on / 12 h off	Local skin reaction
Pregabalin	PHN	75 mg bd → 150–300 mg bd	Renal dose; sedation
Amitriptyline	PHN	10–25 mg nocte → 75 mg	Anticholinergic; cardiac

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Early antiviral therapy shortens acute pain but does not reliably prevent PHN.
- ▸ Live zoster vaccine is contraindicated in immunosuppressed patients.

GUIDELINES & RECENT ADVANCES

- NeuPSIG / Finnerup 2015 neuropathic pain recommendations

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1.1.24 Infections in Patients with Indwelling Devices (Catheters, Stents, Ports)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Device-related infections in palliative care. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Prevent and manage device-related infections (CD4.1, PD8)

DEFINITION Palliative patients often carry urinary catheters, nephrostomies, biliary stents, central lines, ports, gastrostomies and drains — each a portal for infection requiring prevention bundles and pragmatic treatment.

Device infections

Device	Typical infection	Key management
Urinary catheter	CAUTI (bacteriuria common — treat only if symptomatic)	Change catheter, culture, targeted antibiotic
Nephrostomy	Pyelonephritis, tube blockage	Flush/change tube; antibiotics
Central line / port	CRBSI, exit-site and tunnel infection	Paired cultures; remove line for S. aureus, Candida, tunnel infection
Gastrostomy (PEG)	Peristomal cellulitis, abscess	Cleaning, flucloxacillin / co-amoxiclav
Indwelling pleural / peritoneal catheter	Cellulitis, empyema, peritonitis	Antibiotics; catheter often retained

Drugs used — Drug · Indication · Dose · Caution

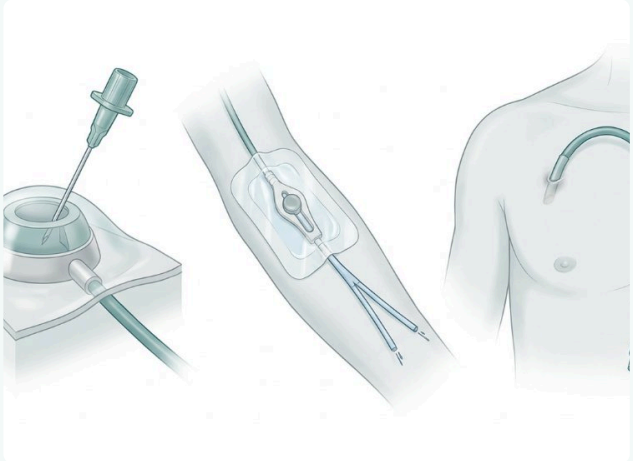
Drug	Indication	Dose	Caution
Nitrofurantoin	Lower UTI (eGFR > 45)	100 mg MR bd × 5 days	Avoid in renal impairment
Vancomycin	Line infection (gram-positive)	15–20 mg/kg IV 12-hourly (levels)	Nephrotoxicity
Flucloxacillin	Exit-site / PEG cellulitis	500 mg–1 g qds	Cholestatic jaundice (rare)

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Asymptomatic bacteriuria in a catheterised patient should not be treated.
- ▸ Review the need for every device at each visit — remove what is no longer useful.

GUIDELINES & RECENT ADVANCES

- IDSA guidelines on CAUTI and CRBSI
- HICPAC device bundles



ILLUSTRATION

Vascular access devices in cancer care

- ▸ Implanted port — access only with a non-coring (Huber) needle; flush after use as per local policy.
- ▸ PICC — inserted through an arm vein; watch for arm swelling (catheter-related DVT) and exit-site redness.
- ▸ Tunnelled CVC — the subcutaneous cuff anchors the line and lowers infection risk.
- ▸ Fever with a line in place: blood cultures from the line and a peripheral vein before antibiotics.

1.1.25 Aspiration Pneumonia in Advanced Illness

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Aspiration pneumonia in patients with dysphagia — prevention and management. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage aspiration risk and pneumonia proportionately (CD4.2, CD6)

DEFINITION Aspiration pneumonia is lung infection after inhalation of oropharyngeal or gastric contents, common with dysphagia from head-and-neck cancer, stroke, dementia, motor neurone disease and reduced consciousness.

Risk factors and prevention

Risk	Prevention
Dysphagia	Speech-and-language assessment; texture modification; upright 30–45° during and after meals
Poor oral hygiene	Twice-daily mouth care, chlorhexidine where appropriate
Sedation	Minimise sedatives at mealtimes
Tube feeding	Does not prevent aspiration of saliva or reflux

Treatment

Setting	Antibiotic
Community / mild	Amoxicillin–clavulanate 625 mg tds × 5–7 days
Hospital / severe	Piperacillin–tazobactam; add anaerobic cover only if abscess / empyema
End of life	Oral antibiotic for symptom relief or comfort care alone

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Amoxicillin–clavulanate	Aspiration pneumonia	625 mg tds oral / 1.2 g IV tds	Diarrhoea, C. difficile
Glycopyrronium	Drooling / sialorrhoea	200–400 µg tds oral	Dry mouth, constipation

★ CLINICAL PEARLS & EXAM FOCUS

- ► 'Risk feeding' with documented acceptance of aspiration risk respects quality of life.
- ► Recurrent aspiration pneumonia is a marker of the last months of life in dementia and MND.

GUIDELINES & RECENT ADVANCES

- BTS Community-Acquired Pneumonia guideline
- RCP 'Supporting people who have eating and drinking difficulties' (2021)

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1.1.26 Influenza, COVID-19 and Respiratory Viral Infections

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Respiratory viral infections in immunocompromised cancer patients. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Prevent and treat respiratory viral infections in palliative patients (CD4.1, CD9)

DEFINITION Respiratory viruses (influenza, SARS-CoV-2, RSV, parainfluenza) cause more severe and prolonged disease in patients with haematological malignancy, lymphopenia, recent chemotherapy or anti-CD20 therapy.

Management

Virus	Treatment	Prevention
Influenza	Oseltamivir 75 mg bd × 5 days (start early)	Annual inactivated vaccine for patients and carers
SARS-CoV-2	Nirmatrelvir–ritonavir (interactions!) or remdesivir in high-risk patients	Vaccination; masks in clinics
RSV	Supportive; ribavirin in selected HSCT recipients	RSV vaccine in older adults where available

Palliative considerations

Issue	Approach
Breathlessness	Opioid, fan (not in infectious settings — use cool flannel), oxygen if hypoxic
Isolation at end of life	Allow family visits with PPE; video calls
Drug interactions	Ritonavir raises levels of fentanyl, oxycodone, midazolam — reduce doses

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Oseltamivir	Influenza	75 mg bd × 5 days (renal adjustment)	Nausea
Nirmatrelvir–ritonavir	COVID-19, high risk	300/100 mg bd × 5 days	Major CYP3A4 interactions

★ CLINICAL PEARLS & EXAM FOCUS

- ► Live vaccines are contraindicated during chemotherapy and immunosuppression.
- ► Avoid fans in infectious respiratory illness (aerosol spread).

GUIDELINES & RECENT ADVANCES

- ECIL-7 / ASCO guidance on respiratory viruses in cancer
- MoHFW clinical guidance for COVID-19

Trauma & Critical care Medicine

1.1.27 Spontaneous and Secondary Bacterial Peritonitis

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Infected ascites in malignancy and cirrhosis — diagnosis and management. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise peritonitis in patients with ascites or peritoneal catheters (CD4.1)

DEFINITION Spontaneous bacterial peritonitis is infection of ascitic fluid without a surgical source (ascitic neutrophils ≥ 250/mm³), chiefly in portal-hypertensive ascites; secondary peritonitis arises from perforation, abscess or peritoneal catheters.

Diagnosis

Feature	SBP	Secondary peritonitis
Neutrophils	≥ 250/mm³	Often > 1000/mm³
Organisms	Single (E. coli, Klebsiella, streptococci)	Polymicrobial, anaerobes
Ascitic glucose / LDH	Normal	Glucose < 50 mg/dL, LDH high, protein > 1 g/dL
Management	Antibiotics + albumin	Imaging; surgical source control if appropriate

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Ceftriaxone	SBP	2 g IV daily × 5 days	—
Human albumin 20%	SBP (renal protection)	1.5 g/kg day 1, 1 g/kg day 3	Fluid overload
Norfloxacin / ciprofloxacin	Secondary prophylaxis	Norfloxacin 400 mg / ciprofloxacin 500 mg daily	Resistance

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Do a diagnostic tap in any patient with ascites who deteriorates.
- ▸ Peritoneal catheter infection rarely needs catheter removal.

GUIDELINES & RECENT ADVANCES

- EASL 2018 guidelines on decompensated cirrhosis

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1.2.1 Hyponatraemia & SIADH in Malignancy

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the causes and management of hyponatraemia in a patient with small cell lung carcinoma. [Paper 2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Diagnose and manage malignancy-associated hyponatraemia including SIADH (CD3.1, PD3)

DEFINITION Hyponatraemia (serum sodium <135 mmol/L) is common in palliative care, caused by SIADH (classically small cell lung carcinoma, but also many drugs including opioids, SSRIs and carbamazepine), poor oral intake, vomiting, or adrenal insufficiency; correction speed must be carefully controlled to avoid osmotic demyelination syndrome.

Cause	Distinguishing Feature
SIADH	Euvolaemic, concentrated urine (urine osmolality >100 mOsm/kg) despite low plasma osmolality, urine sodium >30 mmol/L
Hypovolaemic hyponatraemia (vomiting, diuretics, adrenal insufficiency)	Clinical signs of volume depletion; urine sodium often low unless renal/adrenal cause
Drug-induced (opioids, SSRIs, carbamazepine, chemotherapy)	Temporal relationship to drug initiation; improves on withdrawal

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Tolvaptan	SIADH, symptomatic or resistant to fluid restriction	15 mg orally once daily, titrated	Rapid overcorrection risk – requires close sodium monitoring in a monitored setting at initiation; hepatotoxicity with prolonged use
Hypertonic (3%) Saline	Severe symptomatic hyponatraemia (seizures, severe confusion)	Small-volume bolus per protocol, in a monitored setting	Risk of osmotic demyelination syndrome if sodium is corrected too rapidly (>8-10 mmol/L per 24 hours)

★ CLINICAL PEARLS & EXAM FOCUS

► Correcting chronic hyponatraemia too quickly risks osmotic demyelination syndrome (central pontine myelinolysis) – a frequently tested safety limit of <8-10 mmol/L correction per 24 hours applies.

► Fluid restriction alone is often sufficient first-line management for asymptomatic/mild SIADH-related hyponatraemia before escalating to pharmacological therapy.

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1.2.2 Hypercalcaemia of Malignancy – Pharmacological Management

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the pharmacological management of hypercalcaemia of malignancy. [Paper 1, recurring; see also Section 3 (Pain Management), Algorithm 10 and Case 8]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply pharmacological treatment of hypercalcaemia of malignancy in a structured, stepwise manner (CD3.1, PD3)

DEFINITION Hypercalcaemia of malignancy (corrected serum calcium >10.5 mg/dL) most often results from PTHrP secretion (squamous cancers), osteolytic bone metastases, or rarely ectopic calcitriol production (lymphoma); it is detailed here with a pharmacology focus, complementing the emergency algorithm and case already presented in Section 3 (Pain Management).

Severity	Approach
Mild (<12 mg/dL), asymptomatic	Oral hydration, treat underlying malignancy, monitor
Moderate-severe (≥12 mg/dL) or symptomatic	IV isotonic saline rehydration first, then IV bisphosphonate once rehydrated
Severe, needing rapid control	Add calcitonin for rapid (but short-lived) effect while awaiting bisphosphonate onset (2-4 days)

Drugs used — Drug · Indication · Dose · Caution

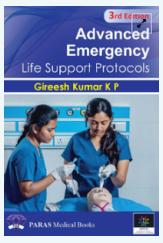
Drug	Indication	Dose	Caution
Isotonic (0.9%) Saline	First-line rehydration, all symptomatic cases	200-300 mL/hour initially, adjusted to clinical/cardiac status	Fluid overload risk in cardiac/renal impairment – monitor closely
Zoledronic Acid	Definitive calcium-lowering therapy	4 mg IV over at least 15 minutes, once rehydrated	Osteonecrosis of jaw, renal impairment, acute phase reaction (fever, myalgia) after first dose
Calcitonin	Rapid short-term effect, severe symptomatic hypercalcaemia	4-8 IU/kg SC/IM every 6-12 hours	Tachyphylaxis after 48-72 hours; nausea

★ CLINICAL PEARLS & EXAM FOCUS

▸ Calcitonin's effect is rapid but wears off within 48-72 hours (tachyphylaxis) – it bridges the gap until the slower-acting but more durable bisphosphonate takes effect, not a substitute for it.

▸ Denosumab is a useful alternative to bisphosphonates in renal impairment, as it does not require renal dose adjustment.

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1.2.3 Hyperkalaemia

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the emergency management of severe hyperkalaemia. [Paper 1, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise ECG changes and manage hyperkalaemia as a medical emergency (CD3.1, PD3)

DEFINITION Hyperkalaemia (serum potassium >5.5 mmol/L) in palliative care arises from renal impairment, tumour lysis syndrome, potassium-sparing drugs (ACE inhibitors, spironolactone), or adrenal insufficiency; severe hyperkalaemia with ECG changes is a medical emergency requiring immediate treatment.

Severity	Management Sequence
Severe with ECG changes (peaked T waves, widened QRS)	IV calcium gluconate immediately to stabilise the cardiac membrane (does not lower potassium itself)
Shift potassium intracellularly	IV insulin with glucose, and/or nebulised salbutamol
Remove potassium from the body	Loop diuretics (if adequate renal function), potassium binders, or dialysis in refractory/severe renal failure cases

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Calcium Gluconate 10%	Cardiac membrane stabilisation, ECG changes present	10 mL IV over 2-3 minutes, repeated if ECG changes persist	Does not lower serum potassium; extravasation causes tissue necrosis
Insulin + Dextrose	Shifts potassium intracellularly	10 units IV insulin with 25 g dextrose IV	Monitor for hypoglycaemia, particularly delayed (1-2 hours later)
Patiromer / Sodium Zirconium Cyclosilicate	Potassium removal, non-emergency/ maintenance	Per product-specific dosing, titrated	Onset too slow for acute emergency management; useful for ongoing/ chronic control

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Calcium gluconate stabilises the myocardial membrane and buys time but does NOT lower the serum potassium – it must always be followed by a potassium-lowering strategy.
- ▸ A haemolysed blood sample is a common cause of spuriously high potassium – always correlate with the ECG and clinical context before treating a surprising result.

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1.2.4 Hypokalaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the causes of hypokalaemia relevant to a palliative care patient. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and manage hypokalaemia in the context of vomiting, diarrhoea and endocrine paraneoplastic syndromes (CD3.1, PD3)

DEFINITION Hypokalaemia (serum potassium <3.5 mmol/L) in palliative care commonly results from vomiting, diarrhoea, diuretic use, or ectopic ACTH secretion (paraneoplastic Cushing syndrome, classically small cell lung carcinoma); severe hypokalaemia risks cardiac arrhythmia and muscle weakness.

Severity	Approach
Mild (3.0-3.5 mmol/L), asymptomatic	Oral potassium supplementation, correct underlying cause
Moderate-severe (<3.0 mmol/L) or symptomatic/ ECG changes	IV potassium replacement under cardiac monitoring, at a controlled infusion rate
Refractory hypokalaemia	Check and correct magnesium – hypomagnesaemia impairs potassium correction

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Potassium Chloride (Oral)	Mild-moderate hypokalaemia	40-100 mmol/day in divided doses	GI upset; avoid in significant renal impairment without monitoring
Potassium Chloride (IV)	Severe/ symptomatic hypokalaemia	Maximum 10-20 mmol/hour via peripheral line (higher rates need central access and cardiac monitoring)	Peripheral vein irritation/ phlebitis; cardiac monitoring required at higher infusion rates

★ CLINICAL PEARLS & EXAM FOCUS

▸ Always check serum magnesium in refractory hypokalaemia – potassium will not correct reliably while magnesium remains low.

EXAM-ORIENTED THEORY

Potassium replacement guide

Serum K (mmol/L)	Replacement
3.0–3.4	Oral KCl 40–80 mmol/day in divided doses
2.5–2.9	Oral + IV; 40 mmol in 1 L saline over 4–6 h
< 2.5 or ECG changes	IV via central line up to 20 mmol/h with cardiac monitoring
Coexisting hypomagnesaemia	Correct Mg — otherwise K stays low
Causes to address	Diarrhoea, vomiting, diuretics, steroids, ectopic ACTH, amphotericin

1.2.5 Hypomagnesaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why does hypomagnesaemia cause refractory hypokalaemia and hypocalcaemia? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise the interrelationship between magnesium and other electrolytes in palliative care (CD3.1)

DEFINITION Hypomagnesaemia (serum magnesium <0.7 mmol/L) is common in palliative care from poor intake, diarrhoea, platinum-based chemotherapy (notably cisplatin), proton pump inhibitor use, and alcohol-related conditions; it causes refractory hypokalaemia and hypocalcaemia if unrecognised.

Cause	Note
Cisplatin nephrotoxicity	Renal magnesium wasting is common and can persist for months after treatment
Chronic diarrhoea/malabsorption	Direct magnesium loss from GI secretions
Proton pump inhibitor use	Impairs intestinal magnesium absorption, particularly with prolonged use

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Magnesium Sulfate (IV)	Symptomatic or severe hypomagnesaemia	2-4 g IV over 1-4 hours (faster for severe symptomatic cases under monitoring)	Rapid infusion can cause hypotension and flushing; caution in renal impairment
Magnesium Oxide/ Glycerophosphate (Oral)	Mild, asymptomatic, or maintenance replacement	Per product-specific dosing, divided through the day	Diarrhoea is the main dose-limiting side effect

★ CLINICAL PEARLS & EXAM FOCUS

► Refractory hypokalaemia or hypocalcaemia that will not correct despite adequate replacement should always prompt checking (and correcting) magnesium first.

EXAM-ORIENTED THEORY

Drugs causing hypomagnesaemia in oncology

Drug	Mechanism
Cisplatin	Renal tubular Mg wasting (can persist for years)
Cetuximab / panitumumab (anti-EGFR)	Blocks Mg reabsorption in the loop of Henle
Proton-pump inhibitors	Reduced intestinal absorption
Amphotericin, aminoglycosides, foscarnet	Tubular toxicity
Diuretics	Renal loss

EXAM-ORIENTED THEORY

Replacement

Severity	Dose
Mild (0.5–0.7 mmol/L)	Oral magnesium (diarrhoea limits dose)
Severe (< 0.5) or symptomatic	MgSO4 8–16 mmol IV over 1–2 h, then 40 mmol over 24 h

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1.2.6 Tumour Lysis Syndrome – Electrolyte Derangements

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the electrolyte abnormalities of tumour lysis syndrome and their management. [Paper 1, recurring; see also Section 4, MCQ on Burkitt lymphoma]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Anticipate, prevent and manage the metabolic derangements of tumour lysis syndrome (CD3.1, CD4.1, PD3)

DEFINITION Tumour lysis syndrome results from the rapid breakdown of a large number of tumour cells (spontaneously or after treatment of a highly chemosensitive, bulky tumour such as Burkitt lymphoma or acute leukaemia), releasing intracellular contents that overwhelm normal excretory/buffering capacity.

Abnormality	Mechanism / Consequence
Hyperkalaemia	Potassium release from lysed cells; risk of cardiac arrhythmia/arrest
Hyperphosphataemia	Phosphate release from lysed cells; can cause secondary hypocalcaemia
Hypocalcaemia	Calcium-phosphate precipitation as phosphate rises; causes tetany, seizures
Hyperuricaemia	Purine breakdown to uric acid; can precipitate in renal tubules causing acute kidney injury

Drugs used — Drug · Indication · Dose · Caution

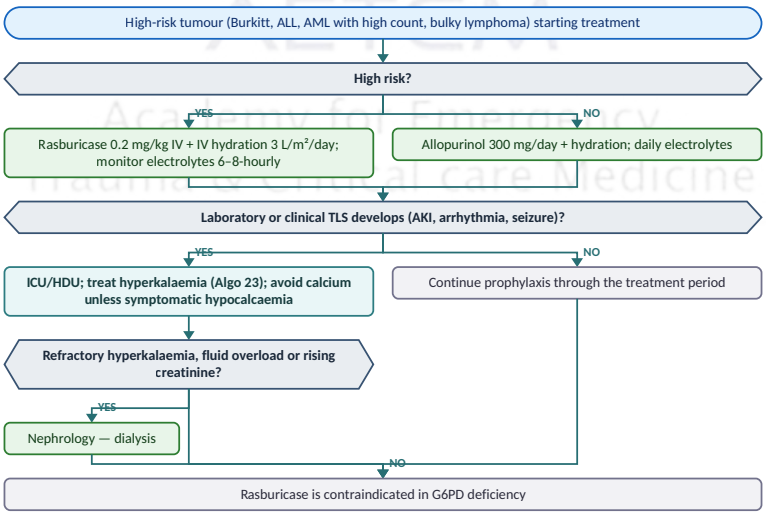
Drug	Indication	Dose	Caution
Rasburicase	High-risk prophylaxis/ treatment of hyperuricaemia	0.2 mg/kg IV once daily for up to 5-7 days	Haemolysis in G6PD deficiency (screen before use) – contraindicated if deficient
Allopurinol	Lower-risk prophylaxis of hyperuricaemia	300 mg orally once daily, started before chemotherapy	Does not reduce existing uric acid, only prevents further production; reduce dose in renal impairment

★ CLINICAL PEARLS & EXAM FOCUS

► Vigorous IV hydration before and during treatment of a bulky, highly chemosensitive tumour is the single most important preventive measure for tumour lysis syndrome.

► G6PD deficiency must be excluded before giving rasburicase, as it can precipitate severe, even fatal, haemolysis in deficient patients.

ALGORITHM · Tumour Lysis Syndrome Algo 18 — full page ›



1.2.7 Refeeding Syndrome

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the pathophysiology and prevention of refeeding syndrome in a severely malnourished palliative care patient. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise patients at risk of refeeding syndrome and institute safe nutritional reintroduction (CD3.1, PD3)

DEFINITION Refeeding syndrome occurs when nutrition (oral, enteral or parenteral) is reintroduced too rapidly in a severely malnourished or cachectic patient, causing a sudden insulin-driven intracellular shift of phosphate, potassium and magnesium, with potentially fatal cardiac and neurological consequences.

Risk Factor	Note
Minimal/no nutritional intake for >7-10 days	Classic high-risk scenario, common in advanced cancer cachexia
Very low BMI or recent significant weight loss	Reflects depleted total body phosphate/magnesium/potassium stores despite normal pre-feeding serum levels
Low baseline serum phosphate, potassium or magnesium before feeding	Predicts a more severe refeeding-related fall once feeding starts

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Thiamine	Prophylaxis, started before/ with refeeding	200-300 mg orally/IV once daily for at least 3 days before and during early refeeding	Essential to prevent Wernicke's encephalopathy, which can be precipitated by carbohydrate refeeding in thiamine-depleted patients
Phosphate, Potassium, Magnesium Replacement	Pre-emptive and reactive correction during refeeding	Per measured levels and local protocol, checked daily initially	Replace proactively rather than only after levels have already fallen

★ CLINICAL PEARLS & EXAM FOCUS

► Start nutrition slowly (typically at a fraction of calculated caloric needs) in at-risk patients, increasing gradually over days while monitoring electrolytes daily – 'start low, go slow' is the governing principle.

► Thiamine should be given before or with the first feed, not after, in any patient at risk of refeeding syndrome or Wernicke's encephalopathy.

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1.2.8 Hypernatraemia and Dehydration

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Hypernatraemia in the dying patient — when to treat? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Assess hydration and sodium disorders in light of goals of care (CD3, CD9)

DEFINITION Hypernatraemia is serum Na > 145 mmol/L, almost always from water deficit exceeding sodium loss — poor oral intake, fever, diarrhoea, osmotic diuresis (hyperglycaemia), diabetes insipidus.

Causes and clues

Type	Causes	Urine osmolality
Pure water loss	Poor intake, fever, tachypnoea	> 600 mOsm/kg
Renal water loss	Diabetes insipidus (pituitary/brain metastases, lithium), osmotic diuresis	< 300 (DI)
Hypotonic fluid loss	Diarrhoea, vomiting, fistula	High

Correction

Step	Detail
Water deficit	0.5 × weight (kg) × (Na/140 − 1)
Rate	Lower Na by ≤ 10 mmol/L per 24 h (chronic)
Fluid	Oral water, 5% dextrose IV, or SC fluids (hypodermoclysis) at home

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
5% dextrose	Water replacement	Rate by deficit; recheck Na 6-hourly	Hyperglycaemia
Desmopressin	Central diabetes insipidus	10 µg intranasal or 100–200 µg oral bd	Hyponatraemia — monitor
0.45% saline SC (hypodermoclysis)	Mild dehydration at home	500–1000 mL/24 h	Local oedema

★ CLINICAL PEARLS & EXAM FOCUS

- ► In the last days of life, thirst is best relieved by mouth care rather than IV fluids.
- ► New polyuria in a patient with brain metastases — think diabetes insipidus.

GUIDELINES & RECENT ADVANCES

- European Hyponatraemia/Hypernatraemia clinical practice guidance
- NICE NG31 — care of dying adults (hydration)

Trauma & Critical care Medicine

1.2.9 Hypocalcaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Causes and management of hypocalcaemia in cancer patients. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and treat hypocalcaemia including drug-induced causes (CD2, CD4.1)

DEFINITION Hypocalcaemia is corrected calcium < 2.1 mmol/L (< 8.5 mg/dL); in cancer it follows denosumab or bisphosphonates (with vitamin D deficiency), tumour lysis, osteoblastic metastases (prostate, breast), hypomagnesaemia and post-thyroidectomy hypoparathyroidism.

Clinical features

System	Features
Neuromuscular	Perioral/fingertip paraesthesia, cramps, tetany, Chvostek and Trousseau signs
Cardiac	Prolonged QTc, arrhythmia, hypotension
CNS	Seizures, confusion

Treatment

Severity	Treatment
Severe / symptomatic / QTc long	10% calcium gluconate 10–20 mL IV over 10 min, then infusion
Mild	Oral calcium 1–2 g/day + cholecalciferol
Hypomagnesaemia	Correct Mg first — calcium will not normalise otherwise

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Calcium gluconate 10%	Symptomatic hypocalcaemia	10–20 mL IV over 10 min; infusion 50 mL in 500 mL over 12–24 h	Extravasation causes necrosis
Calcium carbonate	Maintenance	1–2 g elemental Ca/day in divided doses	Constipation
Calcitriol	Hypoparathyroidism	0.25–1 µg/day	Hypercalciuria

★ CLINICAL PEARLS & EXAM FOCUS

► Always give calcium + vitamin D with denosumab, and check calcium before each dose.

► In TLS, treat only symptomatic hypocalcaemia — calcium–phosphate precipitation risk.

GUIDELINES & RECENT ADVANCES

• Society for Endocrinology emergency guidance: acute hypocalcaemia

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1.2.10 Hypophosphataemia and Hyperphosphataemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Disorders of phosphate in the cancer patient. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Identify and correct phosphate disorders, especially during refeeding (CD3)

DEFINITION Hypophosphataemia (< 0.8 mmol/L) results from refeeding, malnutrition, alcohol, tumour-induced osteomalacia (FGF-23) and drugs; hyperphosphataemia (> 1.5 mmol/L) results from tumour lysis and renal failure.

Comparison

Feature	Hypophosphataemia	Hyperphosphataemia
Causes	Refeeding, starvation, alcohol, FGF-23 tumours, IV iron	TLS, AKI/CKD, rhabdomyolysis
Features	Weakness, respiratory muscle failure, confusion, haemolysis, rhabdomyolysis	Hypocalcaemia (tetany), calcium–phosphate deposition, AKI
Treatment	Oral phosphate; IV if < 0.3 mmol/L or symptomatic	Low-phosphate diet, binders, hydration, dialysis in TLS

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Potassium/sodium phosphate IV	Severe hypophosphataemia	0.3–0.5 mmol/kg over 6–12 h	Hypocalcaemia; do not mix with calcium
Phosphate-Sandoz	Mild–moderate	1–2 tablets bd–tds	Diarrhoea
Sevelamer	Hyperphosphataemia	800 mg tds with meals	GI upset

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Check phosphate before and during nutritional support in the malnourished.
- ▸ Tumour-induced osteomalacia: small mesenchymal tumours secreting FGF-23.

GUIDELINES & RECENT ADVANCES

- • NICE CG32 — refeeding syndrome risk

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1.2.11 Hypermagnesaemia and Magnesium Balance

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Magnesium disorders in palliative care patients. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise iatrogenic magnesium disorders (CD2)

DEFINITION Hypermagnesaemia (Mg > 1.05 mmol/L) is uncommon and mostly iatrogenic — magnesium-containing laxatives or antacids in renal impairment.

Features by level

Mg (mmol/L)	Features
2–3	Nausea, flushing, hypotension, reduced reflexes
3–5	Drowsiness, absent reflexes, bradycardia
> 5	Respiratory paralysis, complete heart block, cardiac arrest

Management

Step	Detail
Stop source	Magnesium hydroxide, magnesium sulphate laxatives, antacids
Antagonise	10% calcium gluconate 10–20 mL IV if severe
Remove	IV fluids + furosemide; dialysis in renal failure

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Calcium gluconate 10%	Severe hypermagnesaemia	10–20 mL IV over 10 min	Monitor ECG
Furosemide	Enhance excretion	20–40 mg IV	Hypovolaemia

★ CLINICAL PEARLS & EXAM FOCUS

- ► Avoid magnesium-based laxatives when eGFR < 30.
- ► Loss of deep tendon reflexes is an early warning sign.

GUIDELINES & RECENT ADVANCES

- Renal Association clinical practice guidelines

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1.2.12 Metabolic Acidosis and Lactic Acidosis in Malignancy

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Lactic acidosis in haematological malignancy — Warburg effect. [Paper 1]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Interpret acid–base disturbances in cancer patients (CD1, CD4.1)

DEFINITION Metabolic acidosis is pH < 7.35 with HCO3 < 22 mmol/L; type B lactic acidosis without hypoperfusion occurs in lymphoma, leukaemia and extensive liver metastases (Warburg effect), and with drugs such as metformin.

Classification by anion gap

Anion gap	Causes in palliative patients
High (> 12)	Lactic (sepsis, shock, type B tumour), ketoacidosis, uraemia, toxins
Normal (hyperchloraemic)	Diarrhoea, fistulas, ureteric diversion (ileal conduit), RTA, saline excess

Management

Cause	Approach
Sepsis / hypoperfusion	Treat source, fluids
Type B tumour-related	Treat malignancy (often heralds poor prognosis); bicarbonate rarely helps
Metformin-associated	Stop metformin; haemodialysis if severe
Symptomatic (Kussmaul breathing)	Opioid for breathlessness when correction not appropriate

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Sodium bicarbonate 8.4%	Severe acidosis pH < 7.1 (selected)	50–100 mmol IV, guided by gases	Fluid/sodium load; hypokalaemia
Sodium bicarbonate tablets	Chronic acidosis (CKD, RTA)	500 mg–1 g tds	Bloating

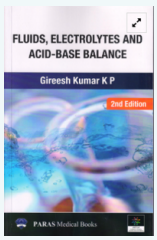
★ CLINICAL PEARLS & EXAM FOCUS

- ► Lactic acidosis in lymphoma is a medical emergency — urgent chemotherapy may reverse it.
- ► Ileal conduit urinary diversion causes hyperchloraemic acidosis.

GUIDELINES & RECENT ADVANCES

- • JBDS guideline on DKA; ESICM acid–base consensus

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1.2.13 Metabolic Alkalosis

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Causes of metabolic alkalosis in palliative patients. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Interpret metabolic alkalosis and its treatment (CD1)

DEFINITION Metabolic alkalosis is pH > 7.45 with HCO3 > 28 mmol/L — commonly from vomiting or nasogastric drainage (loss of HCl), diuretics, steroids/mineralocorticoid excess (ectopic ACTH) and hypokalaemia.

Classification

Type	Urine chloride	Causes	Treatment
Chloride-responsive	< 20 mmol/L	Vomiting, NG drainage, prior diuretics	0.9% saline + KCl
Chloride-resistant	> 20 mmol/L	Ectopic ACTH, hyperaldosteronism, active diuretics, severe hypokalaemia	Treat cause; spironolactone; K replacement

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
0.9% sodium chloride + KCl	Chloride-responsive alkalosis	1–2 L/24 h with 20–40 mmol KCl/L	Fluid overload
Acetazolamide	Alkalosis with fluid overload	250–500 mg daily	Hypokalaemia
PPI	Reduce gastric acid loss with NG drainage	Pantoprazole 40 mg IV daily	—

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Profound hypokalaemic alkalosis + oedema + hyperglycaemia in small-cell lung cancer suggests ectopic ACTH.
- ▸ Alkalosis increases protein-bound calcium — tetany may occur.

GUIDELINES & RECENT ADVANCES

- Seminars in Nephrology — metabolic alkalosis review

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1.2.14 Hypoglycaemia in Malignancy

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Tumour-induced hypoglycaemia — mechanisms and management. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and treat hypoglycaemia, including paraneoplastic causes (CD4.1)

DEFINITION Hypoglycaemia (glucose < 3.9 mmol/L / 70 mg/dL) in cancer arises from insulinoma, non-islet-cell tumour hypoglycaemia (big IGF-2 from large mesenchymal or hepatic tumours), extensive liver metastases, adrenal insufficiency, sepsis, poor intake with continued diabetic drugs.

Causes

Mechanism	Example	Clue
Excess insulin	Insulinoma; sulfonylurea/insulin with poor intake	High insulin and C-peptide
IGF-2 mediated (NICTH)	Large fibrous tumours, hepatoma, sarcoma	Low insulin; raised IGF-2:IGF-1 ratio
Reduced glucose production	Liver replacement by tumour, adrenal failure, sepsis	Low insulin

Management

Step	Detail
Immediate	Glucose 15–20 g oral, or 100 mL 20% dextrose IV, or glucagon 1 mg IM/SC
Prevent	Frequent carbohydrate, continuous dextrose infusion; review diabetic drugs
Specific	Glucocorticoids (prednisolone 20–40 mg) for NICTH; diazoxide/octreotide for insulinoma

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dextrose 20%	Severe hypoglycaemia	100 mL IV, then 10% infusion	Extravasation
Glucagon	No IV access	1 mg IM/SC	Ineffective with depleted glycogen (liver metastases)
Diazoxide	Insulinoma	3–8 mg/kg/day in divided doses	Fluid retention

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ De-intensify diabetic medication as intake falls in advanced illness.
- ▸ Recurrent nocturnal hypoglycaemia with a large abdominal mass → think NICTH.

GUIDELINES & RECENT ADVANCES

- Endocrine Society Clinical Practice Guideline on hypoglycaemia
- Diabetes UK — end-of-life diabetes care

1.2.15 Steroid-Induced Hyperglycaemia and Diabetes at the End of Life

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Management of diabetes in the last months and days of life. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Adapt glycaemic targets to prognosis (CD3, CD8)

DEFINITION Steroid-induced hyperglycaemia is a rise in glucose (classically afternoon/evening) after glucocorticoids; at the end of life the aim shifts from tight control to avoiding symptomatic hyper- and hypoglycaemia.

Targets by prognosis

Stage	Glucose target	Approach
Stable, months–years	6–15 mmol/L	Usual therapy, avoid hypoglycaemia; relax HbA1c
Last weeks	6–15 mmol/L, minimal testing	Simplify: once-daily long-acting insulin
Last days	Check only if symptomatic	Stop oral agents; stop insulin in type 2 if not eating; reduce in type 1

Steroid hyperglycaemia

Situation	Treatment
Morning dexamethasone/prednisolone	Morning gliclazide 40–80 mg or morning NPH insulin 0.1–0.2 U/kg
Steroid dose reduced	Reduce glucose-lowering dose in parallel to avoid hypoglycaemia

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Gliclazide	Steroid hyperglycaemia (morning)	40–80 mg with morning steroid; max 320 mg/day	Hypoglycaemia if intake falls
Isophane (NPH) insulin	Steroid hyperglycaemia	0.1–0.2 units/kg morning, titrate 10–20%	Monitor afternoon glucose
Insulin glargine	Type 1 / insulin-requiring at end of life	Reduce dose by 25%; once daily	Never stop in type 1

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Type 1 diabetes always needs some basal insulin — even when dying — to prevent DKA.
- ▸ Check capillary glucose before the evening meal when steroids are given in the morning.

GUIDELINES & RECENT ADVANCES

- Diabetes UK / TREND: End of Life Diabetes Care (2021)
- JBDS guideline: management of hyperglycaemia and steroid therapy

1.2.16 Adrenal Insufficiency and Adrenal Crisis

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Adrenal crisis in a cancer patient on long-term steroids. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise adrenal crisis, including after immunotherapy and steroid withdrawal (CD4.1, CD2)

DEFINITION Adrenal insufficiency in cancer results from sudden withdrawal of long-term steroids (HPA suppression), bilateral adrenal metastases (lung, breast, melanoma), immune-checkpoint-inhibitor hypophysitis or adrenalitis, and drugs (ketoconazole, mitotane).

Features

Feature	Detail
Symptoms	Fatigue, anorexia, vomiting, abdominal pain, confusion, postural hypotension
Biochemistry	Hyponatraemia, hyperkalaemia (primary only), hypoglycaemia, eosinophilia
Crisis	Shock refractory to fluids
Test	9 am cortisol < 100 nmol/L strongly suggestive; short Synacthen test

Sick-day rules (steroid-dependent)

Situation	Action
Fever / illness	Double usual glucocorticoid dose
Vomiting	IM hydrocortisone 100 mg and seek help
Stopping steroids after > 3 weeks	Taper; do not stop abruptly

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Hydrocortisone	Adrenal crisis	100 mg IV/IM stat, then 50 mg 6-hourly (or 200 mg/24 h infusion)	Hyperglycaemia
0.9% saline	Crisis	1 L over 1 h then per haemodynamics	—
Fludrocortisone	Primary adrenal insufficiency	50–100 µg daily	Oedema, hypokalaemia

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Unexplained deterioration after stopping dexamethasone — consider adrenal insufficiency.
- ▸ Immunotherapy hypophysitis presents with headache and fatigue weeks after starting a checkpoint inhibitor.

GUIDELINES & RECENT ADVANCES

- Society for Endocrinology Adrenal Crisis guidance
- ESMO guideline on immune-related adverse events

1.2.17 Hyperuricaemia and Urate Nephropathy

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Hyperuricaemia in haematological malignancy — prevention and treatment. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Prevent urate nephropathy during cytoreductive therapy (CD4.1)

DEFINITION Hyperuricaemia (urate > 8 mg/dL) follows high cell turnover — leukaemia, lymphoma, myeloproliferative neoplasms — and tumour lysis; urate crystals obstruct renal tubules causing AKI.

Drugs compared

Drug	Mechanism	Use
Allopurinol	Xanthine oxidase inhibitor — prevents new urate	Prophylaxis (low–intermediate risk)
Febuxostat	Non-purine xanthine oxidase inhibitor	Allopurinol intolerance
Rasburicase	Recombinant urate oxidase — degrades existing urate	High risk / established hyperuricaemia

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Allopurinol	Prophylaxis	300 mg/day (100 mg in renal impairment); start 1–2 days before therapy	Rash (HLA-B*58:01), azathioprine interaction
Rasburicase	High risk	0.2 mg/kg IV daily (single dose often sufficient)	Contraindicated in G6PD deficiency; haemolysis
Febuxostat	Allopurinol intolerance	80–120 mg daily	Cardiovascular caution

★ CLINICAL PEARLS & EXAM FOCUS

- ► Samples for urate after rasburicase must be kept on ice (ex-vivo degradation).
- ► Hydration (urine output > 100 mL/h) is the cornerstone of prevention.

GUIDELINES & RECENT ADVANCES

- British Society for Haematology TLS guideline

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1.2.18 Hypoalbuminaemia, Oedema and Fluid Balance in Advanced Cancer

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Peripheral oedema in advanced cancer — causes and management. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Assess and manage oedema and fluid balance appropriately (CD3, CD6)

DEFINITION Hypoalbuminaemia (< 35 g/L) in advanced cancer reflects inflammation (negative acute-phase response), malnutrition, liver disease and protein loss; oedema has multiple causes and needs a cause-specific plan.

Causes of leg oedema in cancer

Cause	Clue	Management
Hypoalbuminaemia	Bilateral, pitting, generalised	Elevation, compression if tolerated; albumin infusion rarely helps
DVT	Unilateral, calf tenderness	Doppler; anticoagulation per goals
Venous / lymphatic obstruction	Pelvic tumour, lymphadenopathy	Dexamethasone trial, radiotherapy, stent; lymphoedema care
Drugs	NSAIDs, steroids, gabapentinoids, calcium-channel blockers	Review medicines
Heart / renal / liver failure	Breathlessness, ascites	Diuretics

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Furosemide	Fluid overload	20–80 mg/day oral or SC infusion	Hypokalaemia, dehydration
Spironolactone	Ascites / hyperaldosteronism	100–400 mg/day	Hyperkalaemia

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Low albumin is a strong adverse prognostic marker (part of the Glasgow Prognostic Score).
- ▸ Diuretics do not help purely hypoalbuminaemic or lymphatic oedema.

GUIDELINES & RECENT ADVANCES

- Modified Glasgow Prognostic Score (mGPS)

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1.2.19 Dehydration and Artificial Hydration at the End of Life

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Clinically assisted hydration in the dying patient — evidence and ethics. [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Make individualised hydration decisions with patients and families (CD8, AD6)

DEFINITION Clinically assisted hydration (IV or subcutaneous) is a medical treatment; in the dying it neither prolongs life nor reliably reduces thirst or delirium, but may help selected patients with reversible dehydration.

Evidence summary

Question	Evidence
Does hydration prolong survival in advanced cancer?	No (Bruera 2013 RCT, 1 L/day vs placebo)
Does it reduce delirium?	No significant benefit in RCT
Can it cause harm?	Oedema, secretions, ascites, effusions, breathlessness

Practical approach

Situation	Approach
Reversible cause (vomiting, diarrhoea, hypercalcaemia)	Time-limited trial of 1 L/24 h SC/IV
Thirst / dry mouth in dying	Mouth care, sips, ice chips
Family requests	Explain, explore cultural meaning; review daily

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
0.9% saline SC (hypodermoclysis)	Mild dehydration	500–1000 mL over 8–24 h	Site oedema; change site every 2–3 days
Hyaluronidase	Improve SC absorption (optional)	150 units into site	Rare allergy

★ CLINICAL PEARLS & EXAM FOCUS

- ► Hypodermoclysis is safe, cheap and suitable for home care in India.
- ► Food and fluid carry deep cultural meaning — involve family in decisions.

GUIDELINES & RECENT ADVANCES

- NICE NG31 Care of dying adults
- Bruera E et al. J Clin Oncol 2013

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1.2.20 Thyroid Dysfunction in Cancer Patients

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Thyroid dysfunction after immunotherapy and radiotherapy. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise endocrine toxicity of cancer treatment (CD4.1, CD2)

DEFINITION Thyroid dysfunction in cancer follows neck radiotherapy, immune-checkpoint inhibitors (thyroiditis with transient thyrotoxicosis then hypothyroidism), tyrosine kinase inhibitors (sunitinib), iodinated contrast and amiodarone.

Patterns

Cause	Typical course	Treatment
Checkpoint inhibitor thyroiditis	Thyrotoxicosis at 4–8 weeks → hypothyroidism	Beta-blocker in thyrotoxic phase; levothyroxine later; continue immunotherapy
Neck radiotherapy	Hypothyroidism months–years later	Levothyroxine
TKIs (sunitinib, lenvatinib)	Hypothyroidism	Levothyroxine
Hypophysitis	Low TSH and low T4 (central)	Hydrocortisone first, then levothyroxine

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Levothyroxine	Hypothyroidism	1.6 µg/kg/day (start 25–50 µg in elderly/cardiac)	Angina, AF
Propranolol	Thyrotoxic symptoms	10–40 mg tds	Asthma

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Fatigue in a patient on immunotherapy — check TSH, free T4 and cortisol.
- ▸ Always replace cortisol before thyroxine in combined deficiency (avoid adrenal crisis).

GUIDELINES & RECENT ADVANCES

- ESMO irAE guideline 2022
- ASCO immune-related adverse events guideline

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1.2.21 Hyperammonaemia (Non-Hepatic)

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Hyperammonaemic encephalopathy without liver failure — causes in cancer patients. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Identify drug-induced metabolic encephalopathy (CD2, CD4.1)

DEFINITION Non-hepatic hyperammonaemia is raised plasma ammonia without significant liver disease, caused by drugs (valproate, 5-fluorouracil, asparaginase, high-dose chemotherapy), urinary tract infection with urease-producing organisms in obstruction, ureterosigmoidostomy and urea-cycle defects.

Causes and management

Cause	Clue	Action
Valproate	Confusion with normal LFTs	Stop valproate; levocarnitine
5-FU / capecitabine	Encephalopathy during infusion	Stop infusion; hydration; lactulose
Asparaginase	ALL treatment	Supportive; dose modification
Urease-producing UTI with obstruction	Proteus, Klebsiella; obstructed urine	Catheterise, antibiotics

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Lactulose	Reduce ammonia absorption	20–30 mL bd–qds	Diarrhoea
Levocarnitine	Valproate hyperammonaemia	100 mg/kg IV loading, then 50 mg/kg 8-hourly	—

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Measure ammonia in an unexplained encephalopathy even if liver tests are normal.
- ▸ Samples must be on ice and analysed promptly.

GUIDELINES & RECENT ADVANCES

- Consensus on valproate-induced hyperammonaemic encephalopathy

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1.2.22 Uraemia and Symptom Control in Renal Failure

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Symptom management in the uraemic patient not for dialysis. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage uraemic symptoms with renally safe prescribing (CD3, CD5)

DEFINITION Uraemia is the clinical syndrome of advanced renal failure — nausea, pruritus, fatigue, breathlessness, restless legs, confusion, myoclonus and pericarditis — that dominates symptom burden in the last weeks of conservative kidney care.

Symptom-specific management

Symptom	First choice	Avoid
Nausea	Haloperidol 0.5–1 mg	Metoclopramide high dose, cyclizine (drowsiness)
Pruritus	Emollients, gabapentin 100 mg alternate days	Antihistamines (little benefit)
Restless legs	Correct iron, gabapentin / pramipexole low dose	—
Pain	Fentanyl, buprenorphine, alfentanil; paracetamol	Morphine, codeine, NSAIDs
Agitation / myoclonus	Midazolam (reduced dose)	Accumulating opioids
Breathlessness (fluid)	Furosemide high dose if passing urine; opioid	—

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Fentanyl (CSCI)	Pain / breathlessness	100–200 µg/24 h starting	Accumulates in prolonged use — monitor
Alfentanil	Pain in eGFR < 15 (CSCI)	0.5–1 mg/24 h starting	Short half-life; specialist
Midazolam	Agitation	5–10 mg/24 h CSCI	Reduce dose in renal failure

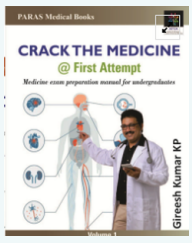
★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Death after stopping dialysis is usually peaceful when symptoms are anticipated.
- ▸ Uraemic myoclonus is often worsened by morphine metabolites.

GUIDELINES & RECENT ADVANCES

- Renal Supportive Care guidance (Kidney Supportive Care Clinical Practice Guideline)

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1.3.1 Venous Thromboembolism in Cancer

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the management of venous thromboembolism in a patient with active malignancy. [Paper 2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Diagnose and manage cancer-associated venous thromboembolism, balancing bleeding and thrombosis risk (CD3.1, PD3)

DEFINITION Cancer confers a 4-7 fold increased risk of venous thromboembolism (DVT/PE) through tumour-related procoagulant factors, immobility, and treatment effects (chemotherapy, central venous catheters); it is a leading cause of death in ambulatory cancer patients receiving chemotherapy.

Scenario	Approach
Confirmed DVT/PE, active cancer	Direct oral anticoagulant (preferred in most patients) or low-molecular-weight heparin, generally continued indefinitely while cancer/treatment is active
GI/genitourinary malignancy with higher bleeding risk	Low-molecular-weight heparin preferred over direct oral anticoagulants given bleeding risk data in luminal GI tumours
Incidental (asymptomatic) PE on staging CT	Treated the same as symptomatic PE in most cases, given comparable prognosis

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Apixaban	First-line oral anticoagulation, most cancer-associated VTE	10 mg twice daily for 7 days, then 5 mg twice daily	Bleeding risk; caution in GI malignancy with intact tumour at risk of bleeding
Low-Molecular-Weight Heparin (e.g. Enoxaparin)	GI/genitourinary malignancy, renal impairment, or where DOACs are contraindicated	1 mg/kg SC twice daily (treatment dose)	Requires daily/near-daily injections; dose-adjust in renal impairment

★ CLINICAL PEARLS & EXAM FOCUS

► Routine prophylactic anticoagulation is not recommended for all ambulatory cancer outpatients, but should be considered in high-risk settings (e.g. pancreatic cancer starting chemotherapy) using a validated risk score (e.g. Khorana score).

► Treatment is generally continued indefinitely while cancer remains active, rather than for a fixed 3-6 month course as in provoked VTE without cancer.

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1.3.2 Anaemia of Chronic Disease & Transfusion Decisions

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the approach to anaemia and blood transfusion decisions in advanced palliative care. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply symptom-based, goals-of-care-aligned transfusion decision-making (CD3.1, PD2, PD3)

DEFINITION Anaemia in advanced cancer results from marrow infiltration, chronic disease-related inflammatory suppression of erythropoiesis, chemotherapy, bleeding, or nutritional deficiency; transfusion decisions in palliative care should be guided by symptoms and goals of care rather than a fixed haemoglobin threshold alone.

Scenario	Consideration
Symptomatic anaemia (fatigue, breathlessness), reasonable prognosis	Red cell transfusion is reasonable for symptom relief, typically targeting symptom improvement rather than a specific number
Asymptomatic, incidentally low haemoglobin	Transfusion generally not indicated purely to 'correct' a number without symptoms
Very limited prognosis (days), transfusion access difficult/burdensome	Weigh the burden of hospital visits/transfusion against uncertain or brief symptomatic benefit – may reasonably decline

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Packed Red Blood Cells	Symptomatic anaemia warranting transfusion	1 unit at a time, reassessed clinically (avoid routinely transfusing 2 units without reassessment)	Transfusion-associated circulatory overload, particularly in frail/ elderly patients – transfuse slowly with diuretic cover if at risk
Erythropoiesis-Stimulating Agents	Chemotherapy-induced anaemia, selected patients	Per product-specific weight/ response-based dosing	Thromboembolism risk, and a theoretical concern about tumour-promoting effect in some cancers – used cautiously and per guideline-restricted indications

★ CLINICAL PEARLS & EXAM FOCUS

► Transfuse one unit at a time and reassess symptoms before giving a second unit – a frequently tested safe-transfusion-practice point, particularly relevant in frail palliative care patients at risk of fluid overload.

► The decision to transfuse (or not) should be revisited as prognosis changes – what is appropriate early in a disease course may not be appropriate in the final days of life.

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1.3.3 Acute Kidney Injury in Advanced Cancer

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the causes of acute kidney injury in a patient with advanced malignancy. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Identify reversible causes of acute kidney injury in cancer and align management with goals of care (CD3.1, PD3)

DEFINITION Acute kidney injury in advanced cancer may be pre-renal (dehydration, sepsis), renal (nephrotoxic chemotherapy, tumour lysis syndrome, myeloma cast nephropathy), or post-renal (ureteric obstruction from pelvic/retroperitoneal tumour or nodal disease) – distinguishing these drives very different management.

Category	Example / Action
Pre-renal	Dehydration from poor intake/vomiting/diarrhoea – cautious rehydration, treat the cause
Renal (intrinsic)	Nephrotoxic chemotherapy (cisplatin), myeloma light-chain cast nephropathy, tumour lysis syndrome – stop/adjust causative drug, treat underlying process
Post-renal (obstructive)	Ureteric obstruction from pelvic/retroperitoneal disease – nephrostomy or ureteric stenting if consistent with goals of care

A GOALS-OF-CARE FRAMEWORK FOR OBSTRUCTIVE UROPATHY

- Relieving obstruction (nephrostomy/stent) can provide meaningful symptom relief and occasionally extend good-quality time, and should not be dismissed purely because the underlying cancer is incurable.
- Equally, in a patient who is actively dying or has expressed a wish to avoid further intervention, allowing progressive renal failure with symptom-focused (uraemic symptom) management is a legitimate and often kinder choice.
- The decision hinges on prognosis, symptom burden, and the patient's own wishes – not on reflexively intervening or reflexively withholding.

★ CLINICAL PEARLS & EXAM FOCUS

► Bilateral ureteric obstruction causing renal failure is a common final pathway in advanced pelvic malignancy (cervical, bladder, prostate, colorectal) – always consider whether decompression is wanted and appropriate before assuming renal failure is simply 'part of dying'.

EXAM-ORIENTED THEORY

AKI in advanced cancer — causes and actions

Cause	Palliative-aware action
Pre-renal (vomiting, poor intake, diuretics, hypercalcaemia)	Fluids if appropriate; stop nephrotoxins
Renal (contrast, cisplatin, NSAIDs, myeloma cast nephropathy, TLS)	Stop offending agents; hydrate; specialist input
Post-renal (ureteric or bladder outlet obstruction)	Catheter; nephrostomy/stent if consistent with goals
Drug dosing	Review opioids (avoid morphine), gabapentinoids, LMWH, antibiotics

1.3.4 Hepatic Failure/Dysfunction in Advanced Cancer

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NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the management of hepatic failure due to extensive liver metastases. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage the consequences of malignant hepatic failure (CD3.1, PD3)

DEFINITION Hepatic dysfunction in advanced cancer results from extensive replacement of liver parenchyma by metastases, primary hepatocellular/cholangiocarcinoma, biliary obstruction, or hepatotoxic chemotherapy; it causes jaundice, coagulopathy, ascites and, in severe cases, hepatic encephalopathy.

Complication	Management Approach
Jaundice from biliary obstruction	Biliary stenting if consistent with goals of care and expected to relieve pruritus/symptoms
Hepatic encephalopathy	Lactulose to reduce ammonia absorption; identify and treat precipitants (infection, GI bleeding, sedatives)
Coagulopathy	Vitamin K trial (limited benefit if synthetic liver failure rather than cholestasis); avoid unnecessary invasive procedures given bleeding risk

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Lactulose	Hepatic encephalopathy	15-30 mL orally 2-3 times daily, titrated to 2-3 soft stools/day	Diarrhoea/bloating if over-dosed; requires adequate fluid intake
Cholestyramine	Pruritus from cholestatic jaundice	4 g orally 1-4 times daily	GI upset, interferes with absorption of other oral medications – separate dosing by several hours

★ CLINICAL PEARLS & EXAM FOCUS

▸ Pruritus from cholestatic jaundice can be severely distressing and disproportionate to the degree of jaundice itself – proactive, specific antipruritic management (not just treating the jaundice) is important.

EXAM-ORIENTED THEORY

Prescribing in liver failure

Drug group	Advice
Opioids	Lower doses, longer intervals; fentanyl / oxycodone with caution; avoid pethidine
Paracetamol	Max 2–3 g/day in hepatic impairment
NSAIDs	Avoid — bleeding, renal failure, ascites
Benzodiazepines	Avoid or use lorazepam (no active metabolites) — encephalopathy risk
Laxatives	Lactulose preferred (also treats encephalopathy)
Capsular pain from liver metastases	Dexamethasone 4–8 mg + NSAID if renal function allows

1.3.5 Disseminated Intravascular Coagulation

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the diagnosis and management of disseminated intravascular coagulation in malignancy. [Paper 1, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage DIC, particularly in acute promyelocytic leukaemia and other malignancies (CD3.1, PD3)

DEFINITION DIC is a consumptive coagulopathy triggered by tissue factor release from tumour cells (classically acute promyelocytic leukaemia, but also adenocarcinomas and sepsis complicating malignancy), causing simultaneous widespread microthrombosis and bleeding as clotting factors and platelets are consumed.

Finding	Significance
Prolonged PT/APTT, low fibrinogen, elevated D-dimer, low platelets	Classic laboratory picture of DIC; a composite score (e.g. ISTH DIC score) aids diagnosis
Bleeding (from venepuncture sites, mucosa, GI tract)	The dominant clinical feature in most solid-tumour-associated and APL-associated DIC
Microvascular thrombosis (less common clinically evident)	Can cause organ dysfunction; more prominent in sepsis-associated DIC

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Fresh Frozen Plasma	Active bleeding with coagulopathy	10-15 mL/kg IV, guided by coagulation parameters and clinical bleeding	Volume overload risk in frail patients; does not address the underlying trigger
Cryoprecipitate	Severe hypofibrinogenaemia with bleeding	Per protocol, targeting fibrinogen >1.5 g/L in active bleeding	Should be guided by repeat fibrinogen measurement
All-trans Retinoic Acid (ATRA)	Suspected/confirmed acute promyelocytic leukaemia	45 mg/m²/day orally in divided doses, started urgently	Differentiation syndrome (fever, breathlessness, fluid retention) – requires prompt steroid treatment if it occurs

★ CLINICAL PEARLS & EXAM FOCUS

► Suspected acute promyelocytic leukaemia with DIC should prompt immediate ATRA initiation even before genetic/cytogenetic confirmation, given the high risk of fatal early haemorrhage if treatment is delayed.

► Treating the underlying trigger (leukaemia induction therapy, sepsis source control) is as important as replacing consumed clotting factors.

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1.3.6 Cancer-Associated Bleeding Diathesis

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the causes and management of bleeding tendency in advanced cancer, distinct from DIC. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Identify and manage non-DIC causes of bleeding in advanced malignancy (CD3.1, PD3)

DEFINITION Beyond DIC, bleeding in advanced cancer can result from thrombocytopenia (marrow infiltration, chemotherapy), tumour erosion into blood vessels, acquired von Willebrand syndrome (extreme thrombocytosis or certain lymphoproliferative disorders), or anticoagulant/antiplatelet therapy given for a separate indication.

Cause	Note
Thrombocytopenia (marrow infiltration/ chemotherapy)	Platelet transfusion threshold depends on bleeding risk and planned procedures, not a single universal number
Tumour erosion into a major vessel (e.g. carotid 'blow-out' in head and neck cancer)	Catastrophic haemorrhage requiring pre-emptive planning and rapid sedation if it occurs (see Section 4, Long-Essay 4)
Concurrent anticoagulation for cancer-associated VTE	Requires ongoing risk-benefit reassessment as bleeding risk evolves through the disease course

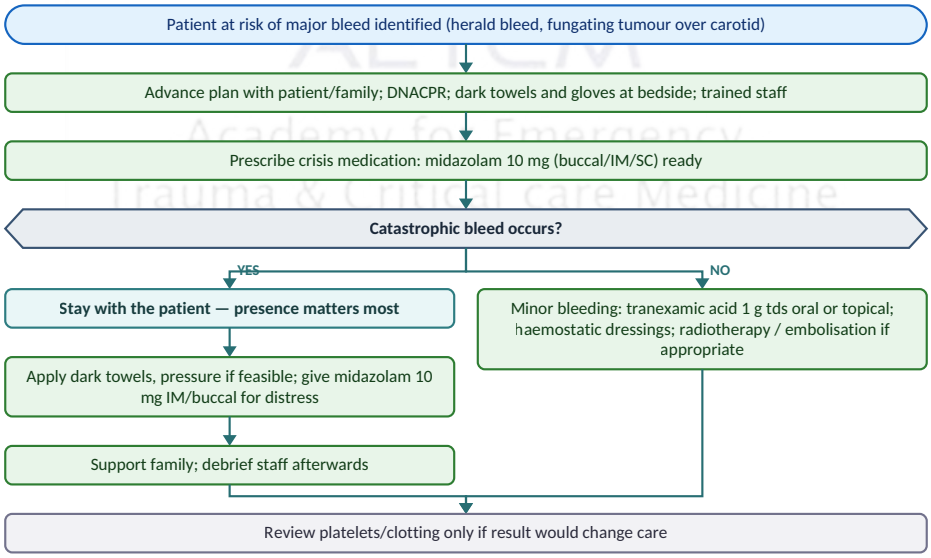
Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Platelet Transfusion	Severe thrombocytopenia with bleeding, or prophylactically before a procedure	1 adult therapeutic dose, reassessed clinically	Alloimmunisation/refractoriness with repeated transfusions; transient benefit only
Tranexamic Acid	Mucosal/surface bleeding (oral, nasal, wound)	500 mg-1 g orally/IV 3 times daily, or topical application to a bleeding surface	Caution in patients at thrombotic risk; generally avoided in frank haematuria due to clot retention risk

★ CLINICAL PEARLS & EXAM FOCUS

► For a patient at known risk of catastrophic bleeding (e.g. carotid erosion in head and neck cancer), pre-emptive planning – dark towels, pre-prepared sedative medication, and prior discussion with the family – is a recognised compassionate palliative measure, discussed further in Section 4.

ALGORITHM · Catastrophic Haemorrhage at the End of Life Algo 20 — full page ›



1.3.7 Pressure Ulcers/Injuries in Palliative Care

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the prevention and management of pressure ulcers in a bedbound palliative care patient. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Prevent, stage and manage pressure injuries in immobile palliative care patients (CD6, PD8)

DEFINITION Pressure ulcers (now termed pressure injuries) result from sustained pressure, shear and friction over bony prominences in immobile, malnourished or oedematous patients; they are largely preventable with structured repositioning and skin-care protocols, and staging guides management once they occur.

Stage	Description
Stage 1	Non-blanchable erythema of intact skin
Stage 2	Partial-thickness skin loss with exposed dermis
Stage 3	Full-thickness skin loss, subcutaneous fat visible, no exposed bone/tendon/muscle
Stage 4	Full-thickness skin and tissue loss with exposed bone, tendon or muscle

PREVENTION AND MANAGEMENT PRINCIPLES

- Regular repositioning (typically every 2 hours if feasible), pressure-redistributing mattresses, and meticulous skin/incontinence care prevent the majority of pressure injuries.
- Adequate nutrition and hydration (where consistent with goals of care) supports tissue integrity and healing capacity.
- In the actively dying patient, some pressure injury (Kennedy terminal ulcer) may be an unavoidable part of the dying process despite optimal care, and family should be counselled accordingly to avoid misplaced guilt.

★ CLINICAL PEARLS & EXAM FOCUS

► Not every pressure injury in a dying patient reflects a failure of care – skin changes at the end of life (Kennedy terminal ulcer) can occur despite best preventive efforts, and families benefit from being told this explicitly.

EXAM-ORIENTED THEORY

Pressure injury staging and care (NPIAP)

Stage	Description	Care
1	Non-blanchable erythema, intact skin	Offload, reposition 2–4-hourly, barrier cream
2	Partial-thickness skin loss	Moist dressing (hydrocolloid/foam)
3	Full-thickness loss, fat visible	Debride slough, foam/alginate, nutrition
4	Bone, tendon or muscle exposed	Specialist wound care; infection control
Unstageable / deep tissue	Base obscured / purple discolouration	Offload; assess as it declares
Kennedy terminal ulcer	Sudden sacral ulcer in the dying	Comfort-focused care; explain to family



ILLUSTRATION

Preventing pressure injury

- Reposition regularly — 30° lateral tilt, pillows between knees and behind the back.
- Float the heels off the mattress; use an alternating-pressure air mattress if high risk.
- Keep skin clean and dry; barrier cream for incontinence; no massage over red areas.
- In the last days, comfort guides turning — not the clock.

1.3.8 Falls & Fractures in Advanced Illness

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the risk factors and prevention of falls in a palliative care patient. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Assess and mitigate fall risk in patients with advanced illness (CD6, PD3)

DEFINITION Falls are common in advanced illness due to weakness, cachexia, sedating medication, postural hypotension, cognitive impairment and bone metastases, and carry particular risk of pathological fracture, head injury (compounded by any coagulopathy), and loss of independence/confidence.

Risk Factor	Mitigation
Sedating medication (opioids, benzodiazepines, antiemetics)	Use the lowest effective dose; review regularly as doses are titrated
Postural hypotension	Review antihypertensives/diuretics; advise slow position changes
Known bone metastases	Mobility aids, physiotherapy input, avoid unsupported weight-bearing on high-risk sites
Cognitive impairment/delirium	Supervised mobilisation, environmental modification (adequate lighting, clutter-free pathways)

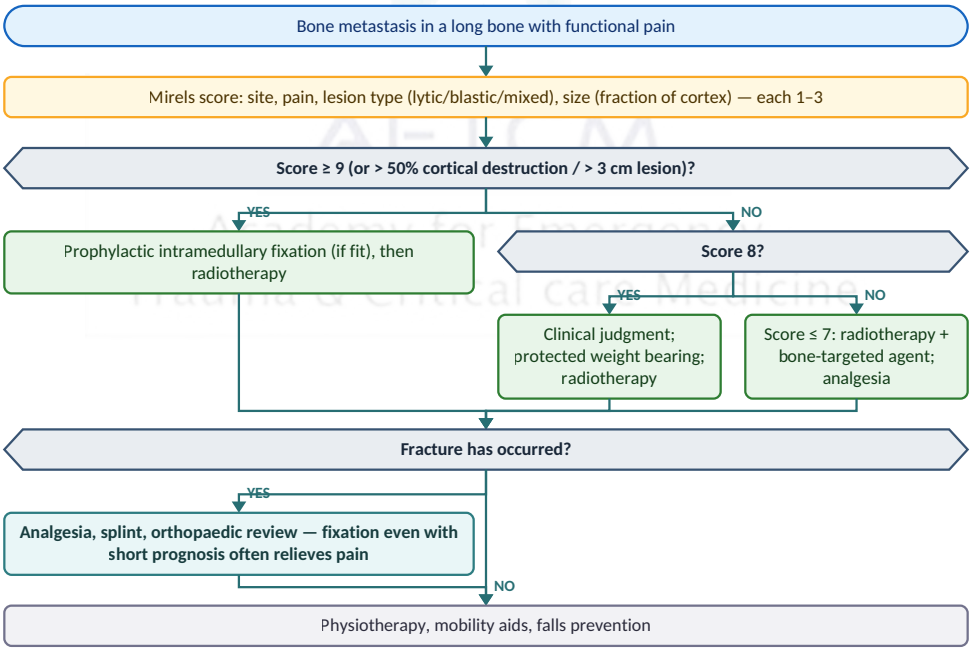
BALANCING SAFETY AND AUTONOMY

- Overly restrictive fall-prevention measures (e.g. physical restraint, excessive bed rest) can themselves cause harm – deconditioning, pressure injury, loss of dignity and autonomy.
- A falls risk assessment should inform a proportionate, individualised plan rather than a blanket restrictive approach, respecting the patient's right to accept some risk in exchange for mobility and independence.

★ CLINICAL PEARLS & EXAM FOCUS

▸ A fall in a patient with known bone metastases should prompt assessment for pathological fracture even if the patient can bear some weight, given metastatic bone is structurally weakened.

ALGORITHM · Impending Pathological Fracture Algo 55 — full page ›



1.3.9 Delirium in the Palliative Care Patient

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the assessment and management of delirium in a hospitalised palliative care patient, distinct from terminal delirium. [Paper 2; see also Section 3 (Pain Management), Algorithm 6]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Assess, investigate and manage delirium using a structured approach across the disease trajectory, not only in the dying phase (CD3.1, CD7, PD3)

DEFINITION Delirium is an acute, fluctuating disturbance of attention and awareness with an underlying medical cause, common at any stage of serious illness (not only in the last days of life, which is covered separately in Section 3 (Pain Management)); it is frequently under-recognised, particularly the hypoactive subtype, which can be mistaken for depression or simple fatigue.

Subtype	Clinical Feature
Hyperactive	Agitation, hallucinations, restlessness – often recognised but sometimes mistaken for anxiety
Hypoactive	Lethargy, withdrawal, reduced responsiveness – frequently missed or mistaken for depression/fatigue
Mixed	Fluctuation between hyperactive and hypoactive features, often within the same day

A STRUCTURED APPROACH (DISTINCT FROM THE TERMINAL DELIRIUM ALGORITHM IN PART II)

- Screen using a validated tool (e.g. 4AT, Confusion Assessment Method) rather than relying on informal impression, given how easily hypoactive delirium is missed.
- Systematically search for and treat reversible causes BEFORE reaching for medication: infection, drug toxicity/withdrawal, metabolic derangement (hypercalcaemia, hyponatraemia), urinary retention, constipation, hypoxia and pain.
- Non-pharmacological measures (reorientation, familiar objects/people, adequate lighting, minimising night-time disturbance) are first-line and should not be skipped even when medication is also used.

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Haloperidol	Pharmacological management of distressing delirium symptoms, after addressing reversible causes	0.5-2 mg orally/SC/IV, repeated as needed	Extrapyramidal side effects at higher doses; caution in Parkinson's disease/Lewy body dementia

★ CLINICAL PEARLS & EXAM FOCUS

▸ Hypoactive delirium is at least as common as hyperactive delirium but far less often recognised – a quiet, withdrawn patient is not necessarily simply 'tired' or 'depressed'.

▸ Delirium earlier in a disease trajectory is often substantially reversible once the precipitant is treated, unlike delirium in the final days of life (terminal delirium, Section 3 (Pain Management)) – this distinction matters for both treatment intensity and prognostic communication.

1.3.10 Immune-Related Adverse Events from Immunotherapy

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the recognition and management of immune-related adverse events from checkpoint inhibitor therapy. [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and grade immune-related adverse events and apply the corticosteroid-based management algorithm (CD2.1, CD4.1, PD3)

DEFINITION Checkpoint inhibitor immunotherapy (anti-PD-1/PD-L1, anti-CTLA-4) can cause immune-related adverse events affecting virtually any organ, by removing the normal brakes on T-cell activation; these can occur at any time during treatment and even months after stopping, and are graded 1-4 by severity to guide management.

Organ System	Example Presentation
GI (colitis)	Diarrhoea, abdominal pain, sometimes bloody stool
Hepatic (hepatitis)	Asymptomatic transaminase rise, or jaundice in severe cases
Pulmonary (pneumonitis)	Cough, breathlessness, new infiltrates on imaging
Endocrine	Thyroiditis (hyper- then hypothyroid), hypophysitis, adrenal insufficiency, type 1 diabetes
Dermatological	Rash, pruritus, vitiligo-like depigmentation

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Prednisone/ Prednisolone (oral)	Grade 2 immune-related adverse events	0.5-1 mg/kg/day, tapered over 4-6 weeks as symptoms resolve	Hyperglycaemia, mood disturbance, infection risk with prolonged use
Methylprednisolone (IV)	Grade 3-4 immune-related adverse events	1-2 mg/kg/day IV, tapered slowly once improving	Higher infection risk at high dose; consider PJP prophylaxis with prolonged high-dose courses
Infliximab	Steroid-refractory colitis specifically	5 mg/kg IV, per specialist protocol	Reactivation of latent tuberculosis/ hepatitis B – screen before use

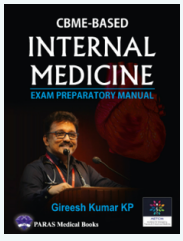
★ CLINICAL PEARLS & EXAM FOCUS

▸ Immune-related adverse events can present months after the last immunotherapy dose – always ask about recent immunotherapy history in any patient with an unexplained new symptom, however distant the last dose.

▸ Endocrine immune-related adverse events (particularly hypophysitis and adrenal insufficiency) are often permanent and require lifelong hormone replacement, unlike most other immune-related adverse events which resolve with steroid treatment.

GUIDELINES & RECENT ADVANCES

- ASCO/ESMO/NCCN Clinical Practice Guidelines: Management of Immune-Related Adverse Events (current version)



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1.3.11 Extravasation Injury from Chemotherapy

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the immediate management of chemotherapy extravasation. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and manage extravasation of vesicant chemotherapy agents (CD2.1, PD8)

DEFINITION Extravasation is the inadvertent leakage of chemotherapy from a vein into surrounding tissue, with vesicant agents (anthracyclines, vinca alkaloids, taxanes) capable of causing severe local tissue necrosis if not managed promptly; irritants cause pain/inflammation without tissue death, and non-vesicants cause minimal local reaction.

Agent Class	Immediate Management
Anthracyclines (e.g. doxorubicin)	Stop infusion immediately, aspirate residual drug, apply cold compress, give dexrazoxane if available/indicated
Vinca alkaloids (e.g. vincristine)	Stop infusion immediately, aspirate residual drug, apply WARM (not cold) compress, consider hyaluronidase
Taxanes	Stop infusion immediately, aspirate residual drug, apply cold compress

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexrazoxane	Anthracycline extravasation, specific antidote	1000 mg/m² IV day 1-2, 500 mg/m² day 3 (within 6 hours of extravasation)	Myelosuppression; must be started promptly to be effective
Hyaluronidase	Vinca alkaloid extravasation, promotes dispersion/ absorption	Injected subcutaneously around the extravasation site per protocol	Local stinging on injection; time-sensitive to be effective

★ CLINICAL PEARLS & EXAM FOCUS

▸ Cold versus warm compress is agent-dependent and easy to mix up – cold for anthracyclines/taxanes (limits spread), warm for vinca alkaloids (promotes dispersion) – a frequently tested, clinically important distinction.

▸ Mark the affected area and photograph it at presentation to monitor progression, and involve plastic surgery early if deep tissue/vesicant extravasation is suspected.

EXAM-ORIENTED THEORY

Extravasation — agent-specific measures

Agent class	Examples	Measure
Anthracyclines	Doxorubicin, epirubicin	Cold packs; dexrazoxane within 6 h
Vinca alkaloids	Vincristine, vinorelbine	Warm packs; hyaluronidase SC
Taxanes	Paclitaxel, docetaxel	Cold (paclitaxel), elevation
Alkylating agents	Mechlorethamine	Sodium thiosulfate
All	Stop infusion, aspirate, mark area, elevate, document, plastic-surgery review if necrosis	

1.3.12 Superior Vena Cava Obstruction

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the clinical features and emergency management of superior vena cava obstruction. [Paper 2, recurring; see also Section 4, Long-Essay 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and urgently manage superior vena cava obstruction as an oncological emergency (CD4.1, PD3)

DEFINITION Superior vena cava obstruction results from extrinsic compression (mediastinal nodal disease, lung cancer, lymphoma) or intraluminal thrombus/tumour invasion of the SVC, impairing venous return from the head, neck and upper limbs; while rarely immediately life-threatening, it can progress to life-threatening airway/ cerebral oedema in rapidly growing tumours.

Feature	Note
Facial/neck swelling, distended neck veins	Classic presenting signs, often worse on waking or bending forward
Breathlessness	From both venous congestion and any associated airway compression
Headache, visual disturbance, altered consciousness	Suggests cerebral oedema – a more urgent presentation requiring prompt treatment

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone	Reduce peritumoural oedema, particularly if lymphoma is suspected (also has antitumour effect in lymphoma)	8-16 mg/day in divided doses	Can obscure lymphoma diagnosis on biopsy if given before tissue is obtained, where biopsy is still planned and feasible

★ CLINICAL PEARLS & EXAM FOCUS

▸ Where tissue diagnosis has not yet been obtained and the patient is not in extremis, try to secure a biopsy before starting corticosteroids, since steroids can rapidly shrink lymphoid tissue and compromise diagnostic yield – balanced against not delaying treatment if the patient is deteriorating rapidly.

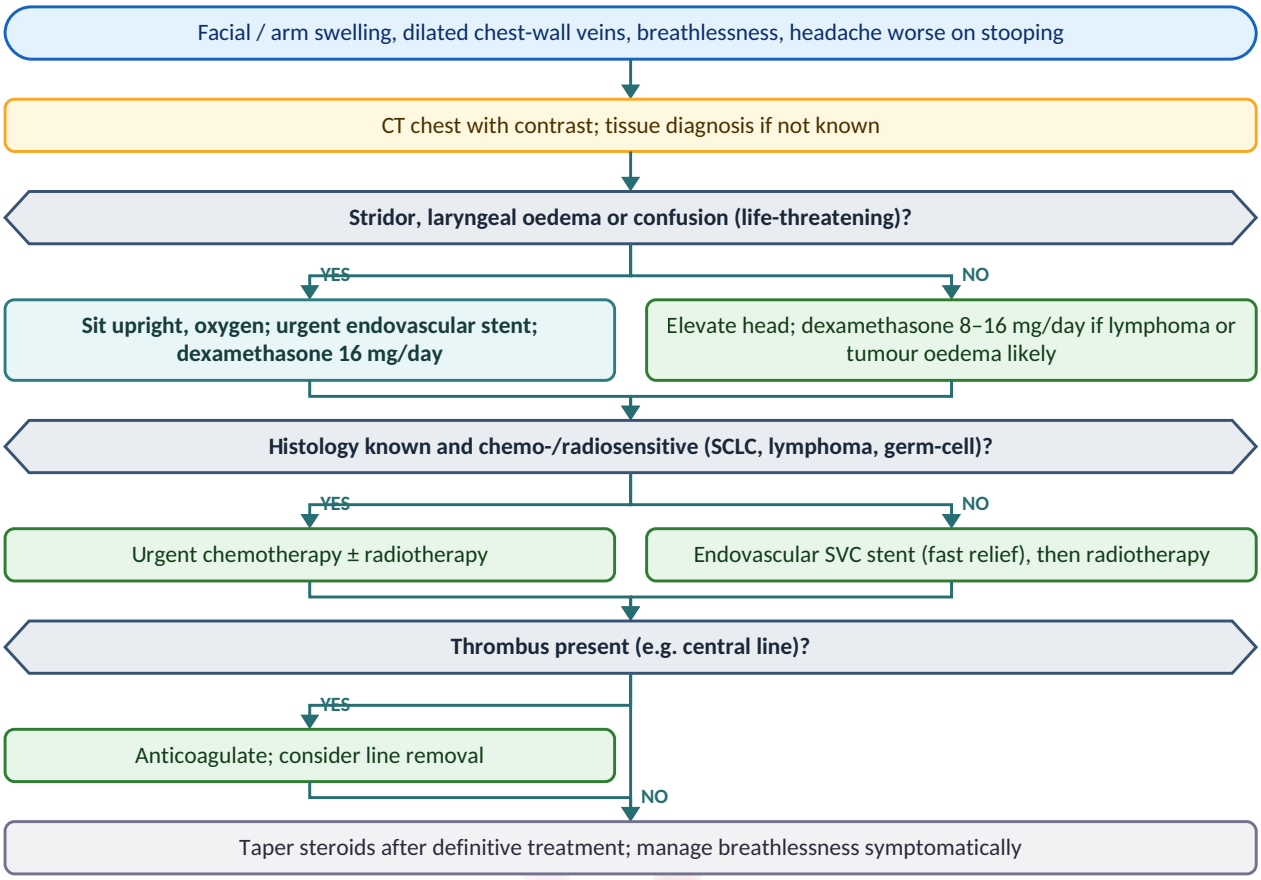
▸ Endovascular stenting of the SVC can provide rapid symptomatic relief and is increasingly used as a bridge to, or alongside, definitive chemotherapy/radiotherapy.

GUIDELINES & RECENT ADVANCES

- See also Section 4, Long-Essay 4 (Oncologic Emergencies) for a comprehensive comparative overview

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ALGORITHM · Superior Vena Cava Obstruction Algo 16 — full page ›



★ **KEY POINTS**

- Obtain histology before steroids if lymphoma is suspected — steroids can mask the diagnosis.
- Stenting gives relief within 24–72 h in > 90%.

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1.3.13 Pulmonary Embolism in the Cancer Patient

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Pulmonary embolism in malignancy — diagnosis, treatment and palliative considerations. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Diagnose and treat PE balancing bleeding risk and prognosis (CD4.1, AD6)

DEFINITION Pulmonary embolism is obstruction of the pulmonary arterial tree by thrombus, usually from deep leg or pelvic veins; cancer increases risk by tumour procoagulants, immobility, surgery, chemotherapy, hormonal therapy and central lines.

Clinical risk and diagnosis

Step	Detail
Symptoms	Sudden breathlessness, pleuritic pain, haemoptysis, syncope, tachycardia, hypoxia
Wells score	> 4: PE likely → CTPA; ≤ 4 → D-dimer (less useful in cancer — often raised)
Imaging	CT pulmonary angiography; V/Q if renal failure; echo for RV strain
Incidental PE	Found on staging CT — treat as symptomatic PE

Treatment by risk

Category	Treatment
High-risk (shock)	Thrombolysis (alteplase 100 mg over 2 h) if goals support; else comfort care
Intermediate / low risk	Anticoagulation for ≥ 6 months and while cancer active
Contraindication to anticoagulation	IVC filter (selected), symptomatic care
Dying	Opioid for breathlessness, anxiolytic; stop anticoagulants

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Enoxaparin	Cancer-associated PE	1 mg/kg SC 12-hourly or 1.5 mg/kg daily	Reduce if CrCl < 30; platelets < 50 000
Apixaban	Cancer-associated VTE (non-GI/GU tumours)	10 mg bd × 7 d, then 5 mg bd	Interactions (azoles, rifampicin); GI bleeding
Rivaroxaban	Alternative DOAC	15 mg bd × 21 d, then 20 mg daily with food	Higher GI/GU bleeding
Alteplase	Massive PE with shock	100 mg IV over 2 h	Major bleeding; brain metastases relative contraindication

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Caravaggio trial: apixaban non-inferior to dalteparin without excess major bleeding.
- ▸ In the dying patient, relief of breathlessness matters more than anticoagulation.

GUIDELINES & RECENT ADVANCES

- ESC 2019 Guidelines on Acute Pulmonary Embolism
- ASCO VTE in cancer guideline update 2023

1.3.14 Deep Vein Thrombosis and Thromboprophylaxis in Palliative Care

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Thromboprophylaxis in palliative care inpatients — when is it appropriate? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Make individualised thromboprophylaxis decisions (CD4.1, AD6)

DEFINITION Deep vein thrombosis is thrombus in the deep veins of the limb or pelvis; Khorana score estimates chemotherapy-associated VTE risk, while prophylaxis in palliative inpatients should match prognosis and goals.

Khorana score

Factor	Points
Very high-risk cancer (stomach, pancreas)	2
High-risk cancer (lung, lymphoma, gynaecological, bladder, testicular)	1
Platelets ≥ 350 000/µL	1
Haemoglobin < 10 g/dL or ESA use	1
Leucocytes > 11 000/µL	1
BMI ≥ 35	1

Prophylaxis decisions

Situation	Recommendation
Khorana ≥ 2, ambulatory on chemotherapy	Consider apixaban 2.5 mg bd / rivaroxaban 10 mg (AVERT, CASSINI)
Inpatient with reversible acute illness	LMWH prophylaxis unless contraindicated
Hospice / last weeks of life	Generally not indicated

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Enoxaparin	Prophylaxis	40 mg SC daily (20 mg if CrCl < 30)	Bleeding, injection burden
Apixaban	Primary prophylaxis (ambulatory, high risk)	2.5 mg bd	Interactions

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ The burden of daily injections matters — ask the patient.
- ▸ Mechanical prophylaxis (stockings) is contraindicated in peripheral arterial disease.

GUIDELINES & RECENT ADVANCES

- ASCO 2023 VTE guideline
- NICE NG89 Venous thromboembolism

1.3.15 Malignant Pericardial Effusion and Cardiac Tamponade

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Cardiac tamponade in malignancy — recognition and management. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise tamponade as an oncological emergency (CD4.1, PD3)

DEFINITION Malignant pericardial effusion occurs with lung and breast cancer, lymphoma and melanoma; tamponade is haemodynamic compromise from raised intrapericardial pressure.

Features

Feature	Detail
Symptoms	Breathlessness, orthopnoea, chest discomfort, fatigue
Signs	Beck's triad (hypotension, raised JVP, muffled heart sounds), pulsus paradoxus > 10 mmHg, tachycardia
ECG / CXR	Low voltage, electrical alternans; globular heart
Echo	Effusion with right atrial/ventricular diastolic collapse

Management

Option	Use
Echo-guided pericardiocentesis	Immediate relief of tamponade
Extended catheter drainage	Reduces recurrence
Pericardial window / sclerosis	Recurrent effusion, longer prognosis
Systemic therapy	Chemosensitive tumours (lymphoma, breast)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
IV fluids	Temporising in tamponade	250–500 mL bolus	Avoid diuretics (reduce preload)

★ CLINICAL PEARLS & EXAM FOCUS

- ► Never give diuretics or vasodilators in tamponade.
- ► Malignant effusion carries a median survival of months — tailor intervention.

GUIDELINES & RECENT ADVANCES

- ESC 2015 Guidelines on Pericardial Diseases

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1.3.16 Hyperviscosity Syndrome

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Hyperviscosity syndrome — causes and emergency management. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and treat hyperviscosity (CD4.1)

DEFINITION Hyperviscosity syndrome is impaired microcirculatory flow from excess paraprotein (Waldenström macroglobulinaemia, myeloma) or cells (leukostasis with WBC > 100 000/µL in acute leukaemia, polycythaemia).

Features and treatment

Feature	Detail
Triad	Mucosal bleeding, visual disturbance (retinal 'sausage' veins, haemorrhages), neurological symptoms (headache, confusion, coma)
Paraprotein type	Plasmapheresis — rapid reduction; then chemotherapy
Leukostasis	Hydroxyurea, leukapheresis, urgent induction; TLS prophylaxis
Avoid	Red-cell transfusion before viscosity is reduced (worsens viscosity)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Hydroxyurea	Leukostasis cytoreduction	50–100 mg/kg/day oral (short term)	Myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

- ► Serum viscosity > 4 cP usually produces symptoms.
- ► Pseudohyperkalaemia and spurious lab results occur with very high counts.

GUIDELINES & RECENT ADVANCES

- IWWM consensus on Waldenström macroglobulinaemia

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1.3.17 Malignant Airway Obstruction and Stridor

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Management of stridor in head-and-neck or lung cancer. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage airway obstruction including end-of-life planning (CD4.1, AD9)

DEFINITION Malignant central airway obstruction is narrowing of the trachea or main bronchi by intraluminal tumour, extrinsic compression or both; stridor indicates > 50–75% luminal narrowing.

Management options

Situation	Intervention
Acute stridor	Sit upright, humidified oxygen, heliox if available, dexamethasone 8–16 mg IV/SC
Intraluminal tumour	Bronchoscopic debulking (laser, cryo, electrocautery)
Extrinsic compression	Airway stent; radiotherapy
Upper airway (larynx)	Tracheostomy if consistent with goals
Not reversible / dying	Opioid + midazolam for distress; calm presence; crisis plan

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone	Tumour oedema	8–16 mg/day	Hyperglycaemia
Midazolam	Terminal distress	2.5–5 mg SC PRN; 10 mg buccal in crisis	Respiratory depression accepted as double effect
Nebulised adrenaline	Temporising	1 mg (1 mL of 1:1000) in 4 mL saline	Tachycardia

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Discuss tracheostomy wishes early in laryngeal cancer — before an emergency.
- ▸ Stridor at rest is an emergency.

GUIDELINES & RECENT ADVANCES

- BTS guideline on interventional bronchoscopy

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1.3.18 Acute Urinary Retention and Ureteric Obstruction

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Obstructive uropathy in pelvic malignancy — palliative options. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage urinary retention and obstructive uropathy aligned to goals (CD4.1, AD6)

DEFINITION Acute urinary retention (bladder outlet obstruction, cord compression, anticholinergic/opioid drugs, constipation) and bilateral ureteric obstruction (cervical, prostate, bladder, colorectal cancer) cause pain, delirium and renal failure.

Approach

Problem	Action
Retention	Bladder scan; catheterise; review drugs (anticholinergics, opioids, TCAs); treat constipation; exclude cord compression
Bilateral hydronephrosis + AKI	Nephrostomy or ureteric stent — weigh benefit, burden, prognosis
Prognosis short / dying	Avoid decompression; symptom care (uraemia is often peaceful)

Decision factors for decompression

Favours	Against
Treatment options remain (chemo/RT)	No further anticancer options
Good performance status	Poor performance status, multiple complications
Patient wishes	Patient declines tubes

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Tamsulosin	Outlet obstruction (prostate)	400 µg daily	Postural hypotension
Oxybutynin	Catheter-related bladder spasm	2.5–5 mg bd–tds	Anticholinergic delirium

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Retention is a reversible cause of terminal agitation — always check the bladder.
- ▸ Nephrostomy in malignant obstruction may add months — but often of poor quality; discuss.

GUIDELINES & RECENT ADVANCES

- EAU guidelines on urinary tract obstruction

Trauma & Critical care Medicine

1.3.19 Hepatic Encephalopathy in Advanced Liver Disease and Liver Metastases

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Hepatic encephalopathy — precipitants and management. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage hepatic encephalopathy and its precipitants (CD4.2, CD3)

DEFINITION Hepatic encephalopathy is reversible neuropsychiatric dysfunction from liver insufficiency or portosystemic shunting; graded West Haven 1 (subtle) to 4 (coma).

Precipitants

Category	Example
GI bleeding	Varices, ulcers
Infection	SBP, UTI, pneumonia
Constipation	Opioids, dehydration
Drugs	Benzodiazepines, opioids, diuretics
Metabolic	Hypokalaemia, hyponatraemia, hypoglycaemia, AKI

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Lactulose	Treatment and prevention	20–30 mL 2–4 times daily to 2–3 soft stools/ day	Diarrhoea, dehydration
Rifaximin	Recurrent encephalopathy	550 mg bd	Cost
Haloperidol (low dose)	Distressing agitation	0.5 mg SC PRN	Avoid benzodiazepines

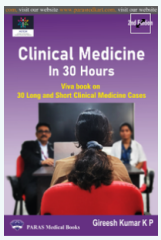
★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Asterixis (flapping tremor) is a key sign but also occurs in uraemia and hypercapnia.
- ▸ In terminal liver failure, the goal is comfort — continue lactulose only if it helps.

GUIDELINES & RECENT ADVANCES

- AASLD/EASL 2014 Hepatic Encephalopathy practice guideline

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1.3.20 Thrombocytopenia and Platelet Transfusion Decisions

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Platelet transfusion thresholds in palliative haematology patients. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Make proportionate transfusion decisions (CD4.1, AD6)

DEFINITION Thrombocytopenia (< 150 000/µL) in cancer follows marrow infiltration, chemotherapy, DIC, sepsis, hypersplenism, drugs (heparin — HIT, linezolid) and immune destruction.

Transfusion thresholds

Situation	Threshold
Prophylactic, stable inpatient	< 10 000/µL
Fever, sepsis, mucositis	< 20 000/µL
Minor procedure / central line	< 20 000–50 000/µL
Active bleeding	< 50 000/µL
Palliative outpatient, chronic	Transfuse for bleeding symptoms, not numbers

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Tranexamic acid	Mucosal bleeding	1 g tds oral	Avoid in haematuria (clot retention)
Platelet transfusion	Bleeding / threshold	1 adult dose (≈ 1 apheresis unit)	Alloimmunisation, febrile reactions

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Refractoriness: check 1-hour post-transfusion increment — < 10 000 suggests immune refractoriness.
- ▸ Transfusion dependence can limit place of care — discuss with patient.

GUIDELINES & RECENT ADVANCES

- BSH guideline on platelet transfusion 2017

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1.3.21 Hypercalcaemia vs Other Causes of Confusion — Approach to Delirium Work-up

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Approach to sudden confusion in an advanced cancer patient. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Perform a proportionate delirium work-up (CD3, CD4.1)

DEFINITION Delirium in cancer is commonly multifactorial; 30–50% of episodes are reversible when the precipitant (drugs, hypercalcaemia, infection, dehydration, hypoxia) is identified and treated.

Reversible precipitants checklist

Category	Check
Drugs	Opioids, benzodiazepines, anticholinergics, steroids, withdrawal (alcohol, nicotine)
Metabolic	Calcium, sodium, glucose, urea/creatinine, liver function
Infection	Urine, chest, lines, wounds
Hypoxia	SpO2
Neurological	Brain metastases, seizures (non-convulsive)
Physical	Urinary retention, constipation, pain

Tools

Tool	Use
4AT	Rapid screening (≥ 4 suggests delirium)
CAM	Diagnostic algorithm
RASS / MDAS	Severity monitoring

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Haloperidol	Distressing delirium	0.5–1 mg oral/SC 2-hourly PRN (max 5 mg/24 h initially)	EPS; avoid in Parkinson's
Olanzapine	Alternative	2.5–5 mg nocte	Sedation

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Investigate only as far as the results would change management.
- ▸ Antipsychotics treat distress, not delirium itself.

GUIDELINES & RECENT ADVANCES

- NICE CG103 Delirium
- Lawlor 2000 — reversibility of delirium in advanced cancer

1.3.22 Acute Pain Crisis (Uncontrolled Severe Pain)

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Management of a cancer pain crisis. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Rapidly titrate opioids safely in a pain emergency (CD3.1, PD3)

DEFINITION A pain crisis is severe, uncontrolled pain (NRS ≥ 7) causing major distress that needs urgent relief within hours.

Rapid titration

Step	Detail
Exclude emergency	Fracture, cord compression, perforation, obstruction, haemorrhage into tumour
IV/SC opioid titration	Opioid-naive: morphine 2–5 mg IV every 15 min until relief; opioid-tolerant: 10–20% of 24-h dose IV/SC every 15–30 min
Then	Convert total titrated dose to regular regimen (CSCI); add adjuvants
Neuropathic / hyperalgesic	Ketamine (specialist), dexamethasone, interventional options

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Morphine IV	Rapid titration	2–5 mg every 15 min (naive)	Monitor RR and sedation
Fentanyl IV	Renal impairment	25–50 µg every 5–10 min	Chest-wall rigidity with rapid large doses

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Stay at the bedside during titration — reassess every 15 minutes.
- ▸ Total pain components amplify crises — address fear and anxiety too.

GUIDELINES & RECENT ADVANCES

- ESMO Clinical Practice Guidelines: management of cancer pain in adults (2018)

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1.3.23 Chemotherapy-Induced Nausea and Vomiting (CINV)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Prevention of chemotherapy-induced nausea and vomiting. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Prescribe antiemetic prophylaxis by emetogenic risk (CD2, CD3)

DEFINITION CINV is nausea/vomiting caused by chemotherapy — acute (< 24 h, 5-HT3 mediated), delayed (24 h–5 days, NK1/substance P mediated) and anticipatory (conditioned).

Prophylaxis by emetogenicity (MASCC/ESMO)

Risk	Example	Prophylaxis
High (> 90%)	Cisplatin, AC (anthracycline + cyclophosphamide)	NK1 antagonist + 5-HT3 antagonist + dexamethasone + olanzapine
Moderate (30–90%)	Carboplatin, oxaliplatin, irinotecan	5-HT3 + dexamethasone (+ NK1 for carboplatin)
Low (10–30%)	Paclitaxel, docetaxel, 5-FU	Single agent (dexamethasone or 5-HT3)
Minimal (< 10%)	Vincristine, bevacizumab	None routinely

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Ondansetron	Acute CINV	8 mg oral/IV before chemo, then 8 mg bd	Constipation, QTc
Aprepitant	Delayed CINV (high risk)	125 mg day 1, 80 mg days 2–3	CYP3A4 interactions; dexamethasone dose ↓
Olanzapine	High-risk / breakthrough CINV	5–10 mg nocte days 1–4	Sedation
Dexamethasone	Acute and delayed	8–12 mg day 1, 8 mg days 2–3	Hyperglycaemia, insomnia

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Anticipatory nausea responds to lorazepam and behavioural therapy — prevention is the best cure.
- ▸ Olanzapine is the most effective rescue agent for breakthrough CINV.

GUIDELINES & RECENT ADVANCES

- MASCC/ESMO Antiemetic Guideline 2023
- ASCO Antiemetics guideline update

Trauma & Critical care Medicine

1.3.24 Chemotherapy-Induced Peripheral Neuropathy (CIPN)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Chemotherapy-induced peripheral neuropathy — agents, features and management. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage treatment-related neuropathic pain (CD3.1, CD4.1)

DEFINITION CIPN is a predominantly sensory, length-dependent ('glove and stocking') neuropathy caused by platinum compounds, taxanes, vinca alkaloids, bortezomib and thalidomide.

Agents

Drug	Features
Oxaliplatin	Acute cold-induced dysaesthesia; chronic sensory neuropathy; 'coasting' after stopping
Cisplatin	Sensory neuropathy, ototoxicity
Paclitaxel / docetaxel	Painful burning feet and hands; acute pain syndrome
Vincristine	Sensorimotor + autonomic (constipation, ileus)
Bortezomib	Painful small-fibre neuropathy; reduced with SC dosing

Management

Step	Detail
Prevention	Dose reduction / delay; no drug proven to prevent (avoid acetyl-L-carnitine)
Painful CIPN	Duloxetine 30–60 mg (best evidence, ASCO)
Other	Gabapentinoids, TCAs, topical menthol/capsaicin; physiotherapy, fall prevention

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Duloxetine	Painful CIPN	30 mg daily × 1 week → 60 mg	Nausea; serotonin syndrome with tramadol
Pregabalin	Alternative	75 mg bd	Sedation, oedema

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Duloxetine is the only agent with RCT support for painful CIPN (Smith 2013).
- ▸ Vincristine neuropathy causes constipation — prescribe laxatives.

GUIDELINES & RECENT ADVANCES

- ASCO guideline: prevention and management of CIPN (2020)

Trauma & Critical care Medicine

1.3.25 Radiation-Induced Complications

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Late effects of radiotherapy relevant to palliative care. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and manage acute and late radiation toxicity (CD4.1)

DEFINITION Radiation toxicity is acute (during and within 90 days: mucositis, dermatitis, oesophagitis, diarrhoea, cystitis) or late (months–years: fibrosis, pneumonitis, proctitis, osteoradionecrosis, lymphoedema).

Common toxicities

Toxicity	Features	Management
Radiation pneumonitis	Cough, breathlessness 1–6 months after thoracic RT	Prednisolone 40–60 mg tapering over weeks
Radiation proctitis	Bleeding, urgency, tenesmus	Sucralfate enemas, formalin, argon plasma coagulation, hyperbaric oxygen
Radiation cystitis	Haematuria, frequency	Bladder irrigation, hyperbaric oxygen
Osteoradionecrosis (jaw)	Exposed bone after head-and-neck RT	Oral care, pentoxifylline–tocopherol, surgery
Pain flare	Transient pain increase after bone RT	Dexamethasone 8 mg prophylaxis

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Prednisolone	Radiation pneumonitis	40–60 mg/day, taper over 6–12 weeks	PJP prophylaxis if prolonged
Sucralfate enema	Radiation proctitis	2 g in 20 mL bd	—

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Pneumonitis is confined to the radiation field — distinguishes it from infection or lymphangitis.
- ▸ Dental assessment before head-and-neck RT prevents osteoradionecrosis.

GUIDELINES & RECENT ADVANCES

- ESTRO / ASTRO consensus on radiation toxicity

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1.3.26 Cardiotoxicity of Cancer Treatment

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Anthracycline cardiotoxicity — risk factors, monitoring and prevention. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise treatment-related heart failure and plan palliative care (CD4.1, CD5)

DEFINITION Cancer therapy-related cardiac dysfunction is a fall in LVEF by ≥ 10% to < 50% (or symptomatic heart failure) caused by anthracyclines (dose-dependent, irreversible), trastuzumab (usually reversible), fluoropyrimidines (vasospasm), immune checkpoint inhibitors (myocarditis) and radiotherapy.

Agents

Agent	Mechanism / feature	Prevention / management
Doxorubicin	Cumulative dose > 450 mg/m² — cardiomyopathy	Dose limits, dexrazoxane, liposomal formulation
Trastuzumab	Reversible LV dysfunction	3-monthly echo; pause and treat
5-FU / capecitabine	Coronary vasospasm (angina)	Stop; nitrates, calcium-channel blocker
Checkpoint inhibitors	Myocarditis (high mortality)	High-dose steroids; stop drug
Mediastinal RT	Late coronary, valve, pericardial disease	Risk-factor control

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
ACE inhibitor (enalapril)	LV dysfunction	2.5 mg bd titrated	Hypotension, renal function
Carvedilol	LV dysfunction	3.125 mg bd titrated	Bradycardia

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Baseline echo before anthracyclines and trastuzumab.
- ▸ End-stage chemotherapy cardiomyopathy needs heart-failure palliative care (see Algo 34).

GUIDELINES & RECENT ADVANCES

- ESC 2022 Cardio-Oncology Guidelines

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1.3.27 Malignant Spinal Cord Compression

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Malignant spinal cord compression — clinical features, imaging and management. [Paper 2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Diagnose and manage MSCC as an oncological emergency (CD4.1, PD3)

DEFINITION Metastatic spinal cord compression is compression of the dural sac and its contents (cord or cauda equina) by extradural tumour, causing actual or impending neurological deficit; commonest with breast, prostate, lung, myeloma and renal cancers.

Clinical features

Stage	Feature
Early	Thoracic or band-like back pain, worse lying flat / coughing, local tenderness
Later	Limb weakness, sensory level, gait ataxia
Late	Bladder / bowel dysfunction, paraplegia
Cauda equina	Saddle anaesthesia, urinary retention, LMN signs

Management summary

Step	Detail
Steroids	Dexamethasone 16 mg/day + PPI
Imaging	Whole-spine MRI within 24 h
Definitive	Surgery + RT (selected) or RT alone (8 Gy single / 20 Gy in 5)
Supportive	Analgesia, VTE prophylaxis, bladder/bowel care, pressure care, rehabilitation
Scoring	Tokuhashi score / SINS for surgical decision

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone	MSCC	16 mg/day oral/IV, taper after definitive therapy	Hyperglycaemia, psychosis, myopathy
Pantoprazole	Gastroprotection	40 mg daily	—

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Ambulatory status at diagnosis predicts ambulatory outcome — speed matters.
- ▸ Educate high-risk patients about warning symptoms (NICE).

GUIDELINES & RECENT ADVANCES

- NICE NG234 Spinal metastases and MSCC (2023)

1.3.28 Malignant Bowel Obstruction

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Malignant bowel obstruction — pathophysiology and management. [Paper 3, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage inoperable bowel obstruction with medical therapy (CD3, CD4.1)

DEFINITION Malignant bowel obstruction is clinical evidence of bowel obstruction beyond the ligament of Treitz in a patient with incurable intra-abdominal cancer or non-intra-abdominal primary with peritoneal disease; common in ovarian (5–50%) and colorectal (10–28%) cancer.

Pathophysiology

Mechanism	Example
Mechanical — extrinsic	Peritoneal deposits, adhesions, mesenteric tumour
Mechanical — intraluminal	Annular or polypoid tumour
Functional (motility)	Infiltration of mesentery/coeliac plexus, opioids, anticholinergics
Secretory cycle	Distension → secretion → further distension (rationale for octreotide)

Medical management

Symptom	Drug
Colic	Hyoscine butylbromide 60–120 mg/24 h CSCI
Continuous pain	Opioid CSCI
Nausea / vomiting	Haloperidol 2.5–5 mg/24 h or cyclizine; levomepromazine
High-volume vomiting	Octreotide 300–600 µg/24 h
Oedema around obstruction	Dexamethasone 8–16 mg trial

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Octreotide	Reduce secretions	300–600 µg/24 h CSCI	Cost; injection-site pain
Hyoscine butylbromide	Colic and secretions	60–120 mg/24 h CSCI	Dry mouth
Dexamethasone	Partial obstruction	8–16 mg SC daily × 5 days, stop if no effect	Hyperglycaemia

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Prokinetics are contraindicated in complete obstruction with colic.
- ▸ Many patients can eat small amounts for pleasure despite obstruction.

GUIDELINES & RECENT ADVANCES

- MASCC MBO guidelines 2021

1.3.29 Raised Intracranial Pressure and Seizures

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Management of raised intracranial pressure in brain metastases. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage neurological emergencies in palliative patients (CD4.1, PD3)

DEFINITION Raised intracranial pressure is elevation of pressure within the skull from tumour mass, vasogenic oedema or hydrocephalus, causing headache (worse on waking, coughing), vomiting, drowsiness and papilloedema; seizures occur in 20–40% of patients with brain metastases.

Management

Problem	Treatment
Vasogenic oedema	Dexamethasone 8–16 mg/day (morning); taper to lowest effective dose
Hydrocephalus	Neurosurgical shunt / ventriculostomy if appropriate
Headache	Paracetamol, opioid; steroid
Vomiting	Cyclizine 50 mg tds (central action)
Seizure prophylaxis	Only after a first seizure (levetiracetam)
Last days	Midazolam CSCI to cover seizures when oral route lost

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone	Cerebral oedema	8–16 mg/day	Hyperglycaemia, myopathy, insomnia
Levetiracetam	Seizures	500 mg bd → 1500 mg bd	Irritability, mood change
Midazolam	Status / end of life	10 mg buccal PRN; 20–30 mg/24 h CSCI	Sedation

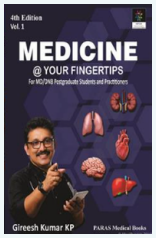
★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Morning headache + vomiting without nausea suggests raised ICP.
- ▸ Avoid lumbar puncture when raised ICP is suspected.

GUIDELINES & RECENT ADVANCES

- EANO–ESMO guideline on brain metastases

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1.3.30 Haemoptysis, Haematuria and Other Bleeding Sites

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Palliative management of bleeding in advanced cancer. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage bleeding with local and systemic measures appropriate to goals (CD4.1, CD3)

DEFINITION Bleeding occurs in 6–10% of patients with advanced cancer — from tumour erosion of vessels, thrombocytopenia, coagulopathy (DIC, liver failure), anticoagulants and treatment (radiation proctitis/cystitis).

Site-specific measures

Site	Local measures	Definitive options
Haemoptysis	Tranexamic acid, cough suppression	Bronchoscopic therapy, embolisation, RT
Haematuria	Three-way catheter with bladder irrigation; alum irrigation	RT, embolisation, cystoscopic diathermy
Rectal / vaginal	Tranexamic acid, sucralfate, packs with adrenaline	RT, embolisation
Skin / fungating	Haemostatic dressings, topical tranexamic acid, adrenaline 1:1000	RT
Upper GI	PPI IV, tranexamic acid	Endoscopy, embolisation

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Tranexamic acid	Mucosal bleeding	1 g tds oral; 500 mg–1 g IV	Avoid in haematuria with clots (risk of clot retention)
Vitamin K	Coagulopathy (liver / warfarin)	10 mg IV	Anaphylactoid reactions (slow IV)

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Dark towels and a crisis plan reduce distress in anticipated major bleeds.
- ▸ Review anticoagulants and antiplatelets in every bleeding patient.

GUIDELINES & RECENT ADVANCES

- Harris DG, Noble SIR. Management of terminal haemorrhage (review)

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1.3.31 Lymphangitis Carcinomatosa and Malignant Breathlessness

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Lymphangitis carcinomatosa — features and palliative management. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage breathlessness from intrathoracic malignancy (CD3, CD4.1)

DEFINITION Lymphangitis carcinomatosa is tumour infiltration of pulmonary lymphatics (breast, lung, stomach, pancreas, prostate) causing progressive breathlessness, dry cough and hypoxia with reticular shadowing and septal thickening on CT.

Causes of breathlessness in cancer

Category	Example	Specific treatment
Airway	Tumour obstruction, stridor	Stent, RT, steroids
Parenchyma	Lymphangitis, metastases, pneumonitis	Steroids; systemic therapy
Pleura / pericardium	Effusion, tamponade	Drainage, pleurodesis, IPC
Vascular	PE, SVCO	Anticoagulation, stent
Systemic	Anaemia, cachexia, anxiety	Transfusion, exercise, CBT

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone	Lymphangitis	8 mg/day trial for 1 week	Steroid side-effects
Morphine (oral IR)	Breathlessness	1–2.5 mg 4-hourly PRN	Constipation
Lorazepam	Panic component	0.5 mg SL PRN	Sedation, falls

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Lymphangitis carries a median survival of weeks to a few months.
- ▸ Opioids are the drug of choice for refractory breathlessness.

GUIDELINES & RECENT ADVANCES

- ATS 2012 statement on dyspnoea; ESMO breathlessness guidance

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1.3.32 Mouth Care, Mucositis and Xerostomia

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NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Oral care in palliative patients — xerostomia and mucositis. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Provide comprehensive mouth care (CD6, CD10)

DEFINITION Mouth problems affect over 80% of patients with advanced cancer — dry mouth (drugs, RT, dehydration, mouth breathing), infection (candida, herpes), mucositis (chemo/RT), ulcers and taste change — and profoundly affect eating, speech and dignity.

Assessment and management

Problem	Management
Dry mouth	Frequent sips, ice chips, saliva substitutes, sugar-free gum, pilocarpine (if gland function)
Coated tongue	Soft toothbrush, sodium bicarbonate rinses
Candidiasis	Nystatin / fluconazole; clean dentures
Mucositis	Benzydamine, lidocaine gel, opioids, photobiomodulation
Taste change	Zinc (limited evidence), flavour enhancement
Dying patient	Moisten lips and mouth 1–2-hourly; lip balm

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Pilocarpine	Radiation xerostomia	5 mg tds	Sweating; avoid in asthma
Benzydamine 0.15%	Mucositis pain	15 mL rinse 1.5–3-hourly	Stinging
Fluconazole	Oral candidiasis	50–100 mg daily × 7–14 days	Interactions

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Good mouth care relieves thirst in the dying better than IV fluids.
- ▸ Teach families to give mouth care — it is a meaningful act of caring.

GUIDELINES & RECENT ADVANCES

- MASCC/ISOO mucositis guidelines 2020

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1.3.33 Cancer Anorexia–Cachexia Syndrome

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PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Define cancer cachexia and discuss its pathophysiology and management. [Paper 1/2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Assess nutritional status and manage cachexia (CD3, CD6)

DEFINITION Cancer cachexia is a multifactorial syndrome of ongoing skeletal muscle loss (with or without fat loss) that cannot be fully reversed by conventional nutrition, leading to progressive functional impairment (Fearon 2011).

Stages (international consensus)

Stage	Criteria	Approach
Pre-cachexia	Weight loss ≤ 5%, anorexia, metabolic change	Monitor, nutrition, exercise
Cachexia	Weight loss > 5% in 6 months, or > 2% with BMI < 20 or sarcopenia	Multimodal therapy
Refractory	Catabolic, unresponsive, PS 3–4, survival < 3 months	Comfort and family support

Pathophysiology

Mediator	Effect
Cytokines (IL-6, TNF-α, IL-1)	Inflammation, anorexia, muscle proteolysis
Tumour factors (PIF, LMF)	Muscle and fat breakdown
Hypothalamic changes	Reduced appetite
Ubiquitin–proteasome pathway	Muscle protein degradation

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Megestrol acetate	Appetite stimulation	160–800 mg/day	VTE, adrenal suppression
Dexamethasone	Short-term appetite	2–4 mg/day × 1–3 weeks	Myopathy
Olanzapine	Appetite / nausea	2.5–5 mg nocte	Sedation

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Weight gain from progestogens is mostly fat and fluid, not muscle.
- ▸ Anamorelin (ghrelin agonist) is approved in Japan.

GUIDELINES & RECENT ADVANCES

- ESMO cachexia guideline 2021
- Fearon K et al. Lancet Oncol 2011

SECTION 2

Common Malignancies

A dedicated chapter – separate from the CBME curriculum reproduction (Section 2) and the Question & Answer practice (Section 4) – opening with a clinical outline of how malignancies cause complications (by local invasion, by metastatic site, as paraneoplastic syndromes, and as treatment toxicity), followed by one hundred common malignancies, one full page each, covering epidemiology, stage-wise treatment, the standard Drug · Indication · Dose · Caution table, and the disease- and treatment-related complications a palliative medicine postgraduate must be able to anticipate and manage.

2.1 Outline: How Malignancies Cause Complications 99

2.2 One Hundred Common Malignancies

AT A GLANCE

Section 2 at a glance — palliative issues by tumour site

Site	Dominant palliative problems
Head and neck	Dysphagia, airway obstruction, catastrophic bleed, fungating wounds
Lung	Breathlessness, haemoptysis, SVCO, brain metastases, hypercalcaemia (squamous), SIADH (small-cell)
Breast	Bone pain, lymphoedema, fungating wound, brain/liver metastases
GI / pancreas	Bowel obstruction, ascites, jaundice, cachexia, coeliac-plexus pain
Gynaecological	Malignant ascites, bowel obstruction, ureteric obstruction, fistulae
Prostate / bladder	Bone pain, cord compression, haematuria, urinary retention
Haematological	Infection, bleeding, transfusion dependence, TLS, hyperviscosity
CNS	Seizures, raised ICP, cognitive and personality change, steroid side-effects

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2.1.1 Epidemiology of Common Malignancies – Overview

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Outline the common malignancies encountered in Indian palliative care practice and their relative burden. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Describe the epidemiology of common malignancies relevant to palliative care practice in India (CD4.1, CD9)

DEFINITION Malignancy complications in a palliative care patient arise through four overlapping mechanisms: direct local invasion of adjacent structures, metastatic spread to specific distant organs, paraneoplastic syndromes mediated by tumour-secreted substances, and toxicity from the treatments given to control the cancer. The following outline pages work through each mechanism before the detailed hundred-malignancy chapter that follows.

Category	Representative Cancers	Approx. Relative Burden In India
Head & neck	Oral cavity, oropharynx, larynx	Among the highest in the world, driven by tobacco chewing/smoking and alcohol
Thoracic	Lung (NSCLC, SCLC), mesothelioma	Rising, strongly linked to smoking and increasingly air pollution
Breast & gynaecological	Breast, cervical, ovarian, endometrial	Breast and cervical cancer together are the leading cancers in Indian women
Gastrointestinal	Oesophageal, gastric, colorectal, hepatobiliary, pancreatic	High regional variation (e.g. oesophageal cancer belt, gallbladder cancer in the Gangetic plain)
Genitourinary	Prostate, bladder, renal	Rising with increasing life expectancy and screening detection
Hepatobiliary	Hepatocellular carcinoma, cholangiocarcinoma, gallbladder	Linked to viral hepatitis, hepatolithiasis and geographic gallbladder cancer clusters
Haematological	Leukaemias, lymphomas, myeloma	A relatively smaller but clinically intensive share, with high symptom and treatment-complication burden
Skin	Melanoma (uncommon), BCC/SCC (common in fair-skinned/high-UV populations)	Lower overall burden in darker-skinned populations but clinically important
CNS	Glioma, meningioma, medulloblastoma (paediatric)	A small overall share with disproportionate neurological symptom burden
Bone & soft tissue	Osteosarcoma, Ewing sarcoma, soft tissue sarcomas	Predominantly affects children/young adults; rare but high-impact
Paediatric	Leukaemia, neuroblastoma, Wilms tumour, retinoblastoma	A distinct spectrum with generally higher cure rates than adult cancers

★ CLINICAL PEARLS & EXAM FOCUS

▸ Knowing the typical complication pattern of a cancer category is often more exam- and practice-relevant than memorising incidence figures alone.

▸ Regional variation in India (e.g. oral cancer belt, gallbladder cancer clusters, oesophageal cancer high-incidence areas) reflects distinct local risk-factor exposures.

GUIDELINES & RECENT ADVANCES

- Indian Council of Medical Research (ICMR) National Cancer Registry Programme, latest report

2.1.2 Local Invasion & Compressive Complications by Site

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the local complications of malignancy according to anatomical site. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Relate anatomical site of malignancy to its characteristic local complications (CD4.1, PD3)

DEFINITION Local invasion complications follow predictably from the anatomy of the primary site: a tumour tends to compress, obstruct, erode or infiltrate the specific structures that lie immediately adjacent to it. Anticipating these site-specific patterns allows earlier recognition and pre-emptive management.

Anatomical Region	Typical Local Complications
Head & neck	Airway obstruction, dysphagia/aspiration, trismus, carotid erosion (catastrophic haemorrhage), cranial nerve palsies
Thorax	Airway obstruction, superior vena cava obstruction, malignant pleural/pericardial effusion, recurrent laryngeal nerve palsy
Abdomen (upper GI/ hepatobiliary)	Biliary obstruction/jaundice, gastric outlet obstruction, GI bleeding, hepatic failure
Abdomen (lower GI/pelvic)	Bowel obstruction, perforation, fistula formation, ureteric obstruction/hydronephrosis
Pelvis (gynaecological/ urological)	Ureteric obstruction, vesicovaginal/rectovaginal fistula, pelvic nerve plexus invasion (severe neuropathic pain)
Bone	Pathological fracture, spinal cord/cauda equina compression, hypercalcaemia
Central nervous system	Raised intracranial pressure, seizures, focal neurological deficit, hydrocephalus

★ CLINICAL PEARLS & EXAM FOCUS

► When a patient with known malignancy develops a new symptom, first ask 'what structures lie next to the primary site or known metastases?' before assuming disease progression is diffuse.

EXAM-ORIENTED THEORY

Compressive complications by site

Site	Complication	Palliative intervention
Mediastinum	SVCO, tracheal compression	Stent, radiotherapy, steroids
Spine	Cord compression	Steroids, RT, surgery
Pelvis	Ureteric obstruction, lymphoedema, plexopathy	Stents/nephrostomy, compression therapy, nerve blocks
Biliary tree	Obstructive jaundice	ERCP / PTBD stent
Bowel	Obstruction	Stent, surgery, medical management
Brachial / lumbosacral plexus	Neuropathic pain, weakness	Adjuvants, RT, nerve block

2.1.3 Metastatic Complications – Bone

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the complications of bone metastases and their management. [Paper 3, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage bone metastasis-related pain, fracture risk and hypercalcaemia (CD3.1, PD3)

DEFINITION Bone is among the commonest sites of metastasis (breast, prostate, lung, renal, thyroid, multiple myeloma) and a leading cause of pain and functional impairment in palliative care.

Complication	Mechanism / Note
Pain	Lytic/mixed lesions cause nociceptive bone pain, often worse at night and with weight-bearing
Pathological fracture	Weakened cortical bone fails under normal physiological load; vertebral bodies and long-bone metaphyses are classic sites
Hypercalcaemia	Osteoclast activation releases calcium; managed with rehydration and bisphosphonates
Spinal cord/cauda equina compression	Vertebral metastases extend into the epidural space; an oncological emergency requiring immediate corticosteroids and urgent imaging
Bone marrow infiltration	Diffuse skeletal metastases can cause cytopenias (leucoerythroblastic picture) mimicking primary haematological disease

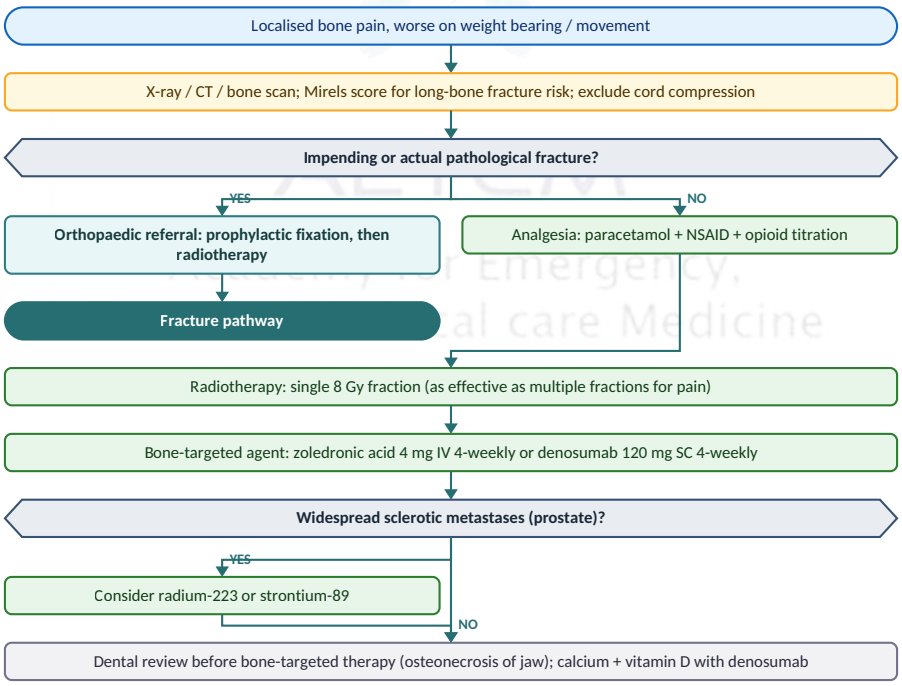
★ CLINICAL PEARLS & EXAM FOCUS

▸ Mirels' scoring helps decide whether prophylactic surgical fixation is warranted before a pathological fracture occurs.

GUIDELINES & RECENT ADVANCES

- See also Section 3 (Pain Management), Algorithm 9 and 10 (cord compression, hypercalcaemia) for emergency management pathways

ALGORITHM · Pain from Bone Metastases Algo 26 — full page ›



2.1.4 Metastatic Complications – Liver

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the complications of hepatic metastases. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and manage hepatic metastasis-related complications (CD3.1, CD5)

DEFINITION The liver is the commonest site of metastasis for colorectal, pancreatic, gastric, breast and neuroendocrine primaries, given portal venous drainage from the GI tract and the liver's general role as a metastatic filter.

Complication	Mechanism / Note
Hepatomegaly & right upper quadrant pain	Capsular stretch from replacement of liver parenchyma by tumour deposits
Jaundice	Either diffuse parenchymal replacement or extrinsic/intrinsic biliary obstruction by hilar deposits
Hepatic failure	Extensive replacement of functioning liver tissue; ascites, encephalopathy and coagulopathy follow
Ascites	Peritoneal spread or portal venous/lymphatic obstruction by liver metastases
Hypoglycaemia (uncommon)	Massive hepatic tumour burden can occasionally cause paraneoplastic hypoglycaemia

★ CLINICAL PEARLS & EXAM FOCUS

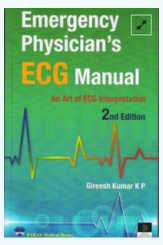
▸ Deranged liver function tests in a patient with known cancer should prompt imaging for hepatic metastases even without overt symptoms.

EXAM-ORIENTED THEORY

Symptoms of liver metastases and their control

Symptom	Management
Right upper quadrant (capsular) pain	Dexamethasone 4–8 mg, NSAID, opioid; coeliac block if refractory
Referred right shoulder-tip pain	As above
Jaundice / pruritus	Biliary stent if obstructive; see pruritus algorithm
Ascites	Paracentesis; spironolactone if SAAG high
Hepatic encephalopathy	Lactulose; avoid sedatives
Hypoglycaemia	Frequent feeds; dextrose

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2.1.5 Metastatic Complications – Lung & Pleura

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the causes and management of breathlessness due to pulmonary metastases. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage pulmonary and pleural metastatic complications causing breathlessness (CD3.1, PD3)

DEFINITION The lung is a common metastatic site for almost all solid tumours given its role as the first capillary bed encountered by venous blood-borne tumour emboli.

Complication	Mechanism / Note
Malignant pleural effusion	Impaired pleural fluid resorption from pleural tumour deposits or lymphatic obstruction; causes breathlessness
Lymphangitis carcinomatosa	Diffuse tumour infiltration of pulmonary lymphatics; causes progressive, often severe breathlessness with a characteristic radiological pattern
Endobronchial obstruction	Causes post-obstructive pneumonia, atelectasis or haemoptysis
Pulmonary nodules/miliary spread	Often asymptomatic until extensive, then progressive breathlessness and hypoxia

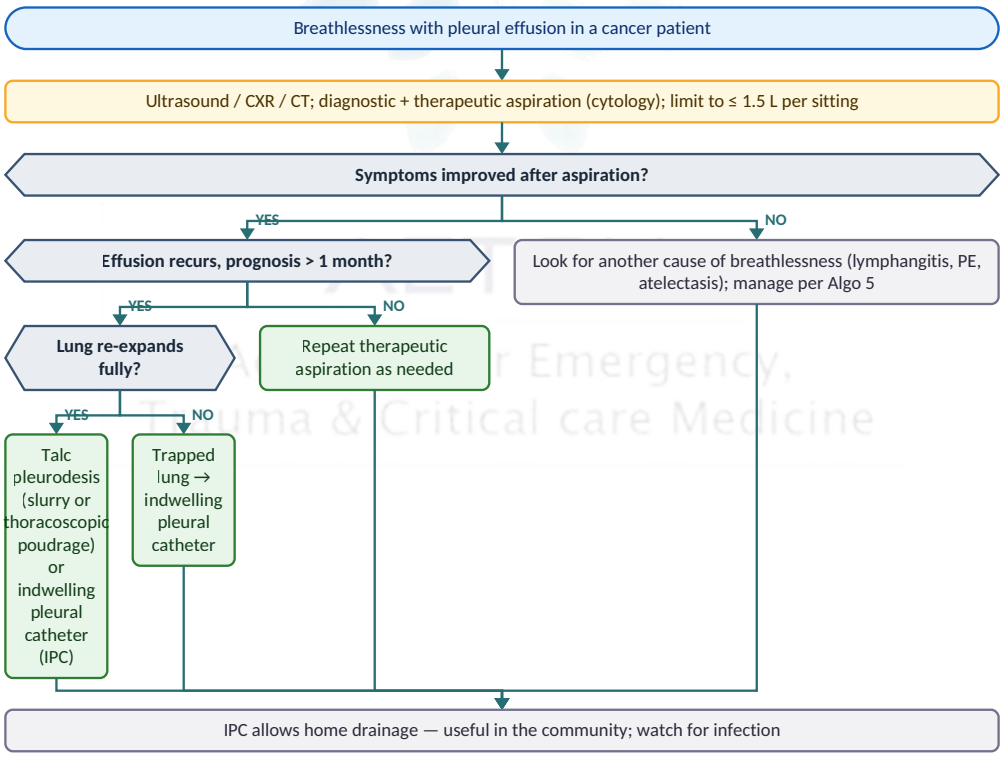
★ CLINICAL PEARLS & EXAM FOCUS

► Lymphangitis carcinomatosa often causes severe breathlessness out of proportion to apparently modest chest X-ray findings – a high-resolution CT appearance is more typical and diagnostic.

GUIDELINES & RECENT ADVANCES

- See also Section 3 (Pain Management), Algorithm 5 and 11 for structured approaches to breathlessness and malignant pleural effusion

ALGORITHM · Approach to Malignant Pleural Effusion Algo 11 — full page ›



2.1.6 Metastatic Complications – Brain

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the clinical presentation and management of brain metastases. [Paper 3, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage brain metastasis-related neurological complications (CD3.1, PD3)

DEFINITION Brain metastases arise most often from lung, breast, melanoma, renal cell and colorectal primaries, and represent a distinct management problem from the primary cancer, often dominating the clinical picture and prognosis.

Complication	Mechanism / Note
Raised intracranial pressure	Mass effect ± surrounding vasogenic oedema; causes headache, vomiting, declining consciousness
Seizures	Cortical irritation from the metastasis or surrounding oedema
Focal neurological deficit	Depends on location – hemiparesis, aphasia, visual field loss, ataxia
Haemorrhage into a metastasis	Particularly melanoma, renal cell carcinoma, choriocarcinoma and thyroid cancer
Leptomeningeal metastasis	Diffuse CSF-based spread causing multifocal cranial neuropathies, headache and hydrocephalus; generally a poor prognostic development

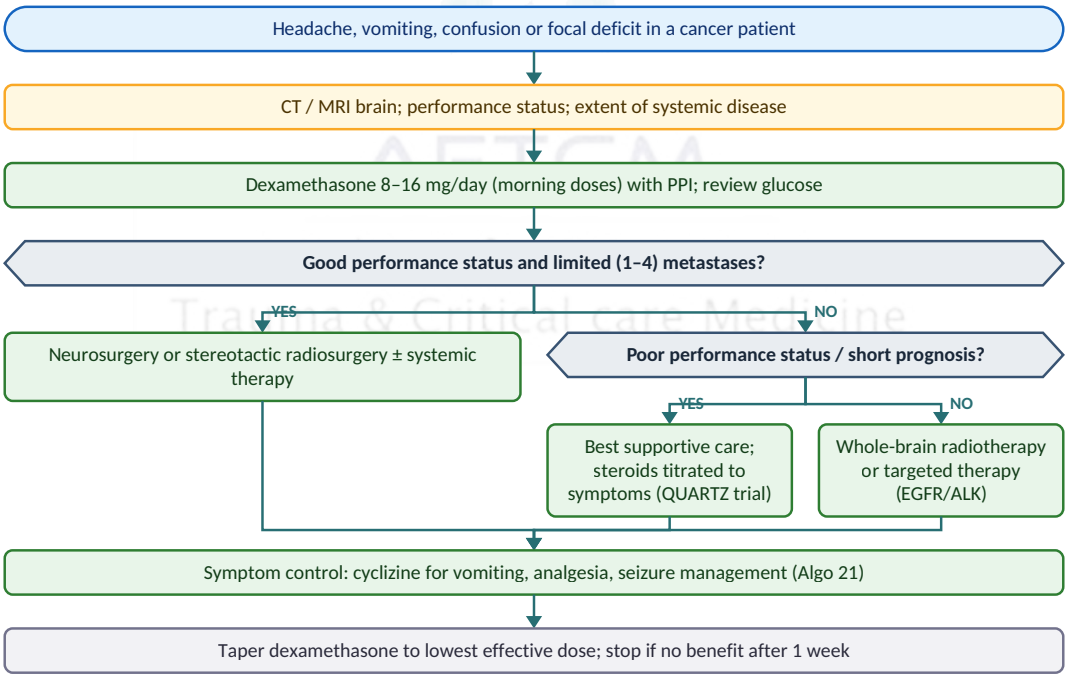
★ CLINICAL PEARLS & EXAM FOCUS

▸ New headache with vomiting, especially worse in the morning or with straining, in a patient with known cancer warrants urgent brain imaging.

GUIDELINES & RECENT ADVANCES

- See Malignancy M80 (Brain Metastases) for detailed management, and Section 3 (Pain Management) Algorithm 6 for terminal delirium which can mimic or coexist with CNS metastatic disease

ALGORITHM · Raised Intracranial Pressure and Brain Metastases Algo 22 — full page ›



2.1.7 Metastatic Complications – Peritoneum

Practice Qs › Algorithm › NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the management of malignant ascites and peritoneal carcinomatosis. [Paper 3] Answer ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage peritoneal metastatic complications including malignant ascites (CD3.1, PD3)

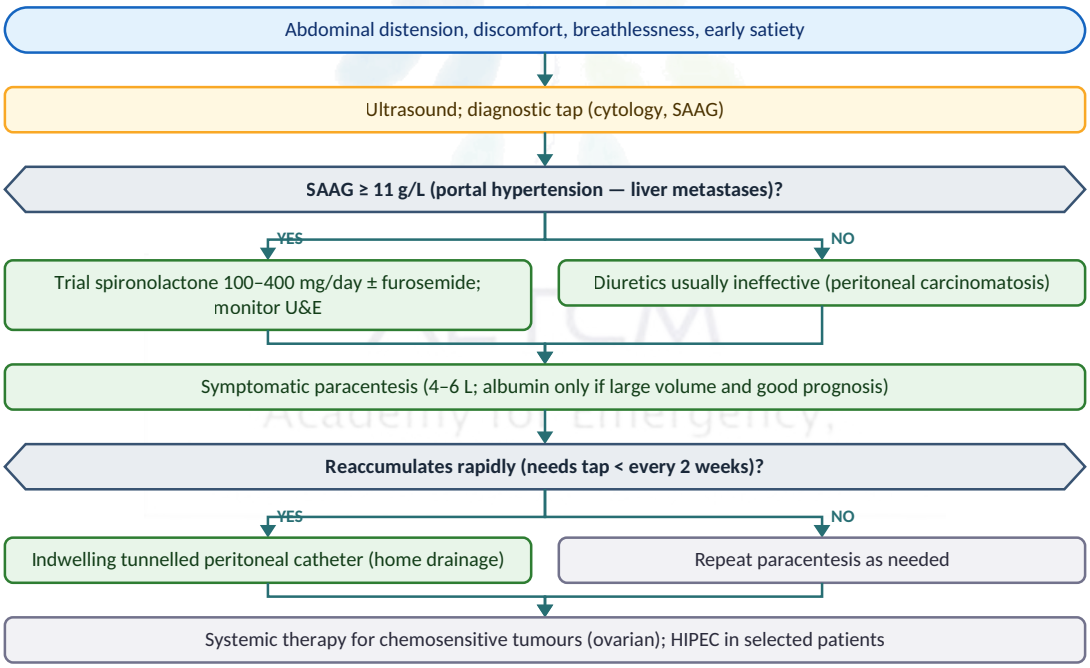
DEFINITION Peritoneal spread is characteristic of ovarian, gastric, appendiceal and colorectal cancers, and produces a distinct complication pattern dominated by fluid accumulation and diffuse bowel involvement rather than a single discrete mass effect.

Complication	Mechanism / Note
Malignant ascites	Peritoneal carcinomatosis increases fluid production and impairs lymphatic drainage; causes distension, breathlessness, early satiety
Bowel obstruction	Diffuse peritoneal deposits can cause multifocal, sometimes intermittent obstruction distinct from a single mechanical blockage
Omental caking	Diffuse omental tumour infiltration, classically seen in ovarian and gastric cancer
Hydronephrosis	Peritoneal/retroperitoneal disease can obstruct the ureters bilaterally, causing renal impairment

★ CLINICAL PEARLS & EXAM FOCUS

▸ Malignant bowel obstruction from peritoneal carcinomatosis is often multifocal and may not be amenable to a single surgical correction – medical management (per Section 3 (Pain Management), Algorithm 4) is frequently the mainstay.

ALGORITHM · Malignant Ascites Algo 41 — full page ›



★ KEY POINTS

▸ Malignant ascites median survival is often < 6 months — discuss goals.

▸ Catumaxomab (EpCAM) was used in Europe for EpCAM+ ascites.

2.1.8 Paraneoplastic Syndromes – Endocrine & Metabolic

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q List the endocrine paraneoplastic syndromes and their associated malignancies. [Paper 2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise endocrine paraneoplastic syndromes and their causative malignancies (CD4.1, CD9.1)

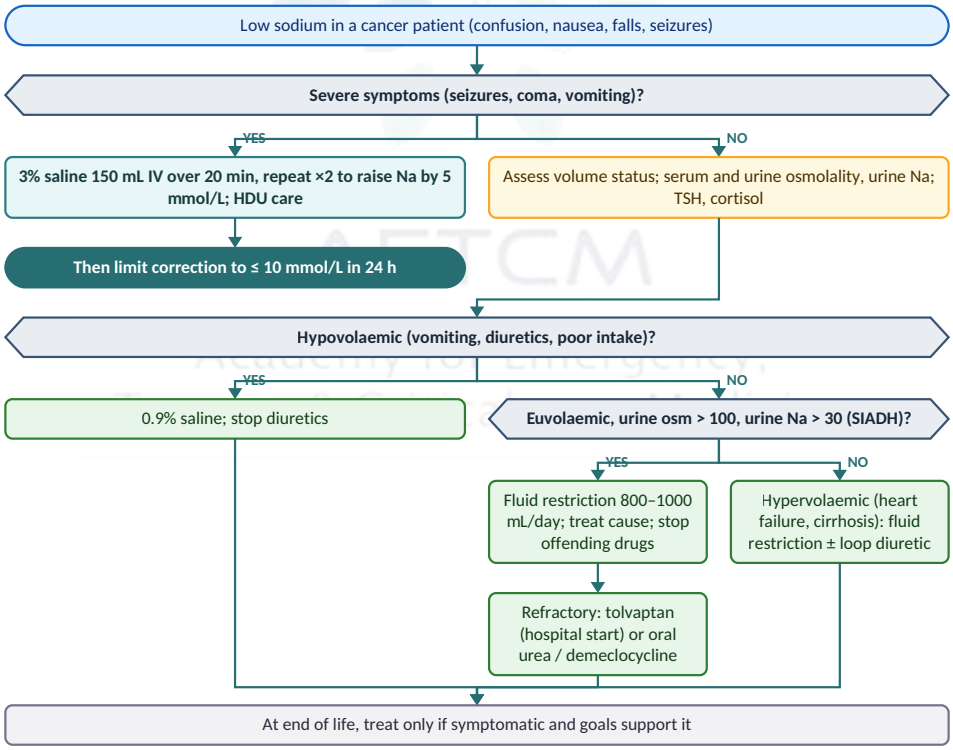
DEFINITION Paraneoplastic syndromes result from substances secreted by the tumour (hormones, peptides, antibodies) acting at a distance from the tumour itself, and can be the presenting feature of an otherwise occult malignancy.

Syndrome	Mechanism / Classic Association
Hypercalcaemia of malignancy	PTHrP secretion (squamous cancers), osteolytic bone metastases, or rarely ectopic calcitriol production (lymphoma)
SIADH	Ectopic ADH secretion, classically small cell lung carcinoma; causes hyponatraemia
Ectopic Cushing syndrome	Ectopic ACTH secretion, classically small cell lung carcinoma and carcinoid tumours; causes hypokalaemia and hyperglycaemia
Hypoglycaemia	Non-islet cell tumour hypoglycaemia from IGF-2 secretion, seen with large mesenchymal/ hepatic tumours
Carcinoid syndrome	Serotonin and other vasoactive peptide secretion once hepatic first-pass metabolism is bypassed (liver metastases)

★ CLINICAL PEARLS & EXAM FOCUS

► A paraneoplastic syndrome occasionally presents before the primary tumour is detectable – persistent, unexplained metabolic derangement in an at-risk patient warrants a search for occult malignancy.

ALGORITHM · Hyponatraemia and SIADH in Malignancy Algo 17 — full page ›



2.1.9 Paraneoplastic Syndromes – Neurological, Dermatological & Haematological

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q List the neurological paraneoplastic syndromes and their associated malignancies. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise non-endocrine paraneoplastic syndromes relevant to palliative oncology (CD4.1, CD9.1)

DEFINITION Beyond endocrine effects, tumours can trigger paraneoplastic neurological (often antibody-mediated), dermatological and haematological syndromes, many of which are more disabling than the underlying tumour burden itself.

Syndrome	Mechanism / Classic Association
Lambert-Eaton myasthenic syndrome	Antibodies against presynaptic voltage-gated calcium channels; proximal weakness improving with repeated effort; associated with small cell lung carcinoma
Paraneoplastic cerebellar degeneration	Anti-Yo, anti-Hu and related antibodies; associated with ovarian, breast and lung cancer
Opsoclonus-myoclonus-ataxia syndrome	Presumed autoimmune; classically associated with neuroblastoma in children
Encephalomyelitis (anti-Hu)	Associated with small cell lung carcinoma; can precede cancer diagnosis by months

Dermatological

Syndrome	Mechanism / Classic Association
Acanthosis nigricans (malignant)	Velvety hyperpigmented skin, classically associated with gastric adenocarcinoma
Dermatomyositis	Proximal myopathy with characteristic skin findings (heliotrope rash, Gottron papules); associated with ovarian, lung and GI malignancy, particularly in older patients
Necrolytic migratory erythema	Associated with glucagonoma

Haematological

Syndrome	Mechanism / Classic Association
Trousseau syndrome (migratory thrombophlebitis)	Hypercoagulable state classically associated with pancreatic adenocarcinoma
Non-bacterial thrombotic (marantic) endocarditis	Sterile valvular vegetations causing embolic events in advanced malignancy
Paraneoplastic erythrocytosis	Ectopic erythropoietin secretion, classically renal cell carcinoma

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★ CLINICAL PEARLS & EXAM FOCUS

► Paraneoplastic neurological syndromes are often irreversible even with successful cancer treatment, since antibody-mediated neuronal damage may not recover – early recognition and treatment gives the best chance of a response.

2.1.10 Treatment-Related Complications – Chemotherapy Toxicity Classes

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Classify the major toxicities of cytotoxic chemotherapy with examples. [Paper 1, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Anticipate and manage chemotherapy toxicity classes relevant to palliative/supportive care (CD2.1, PD3)

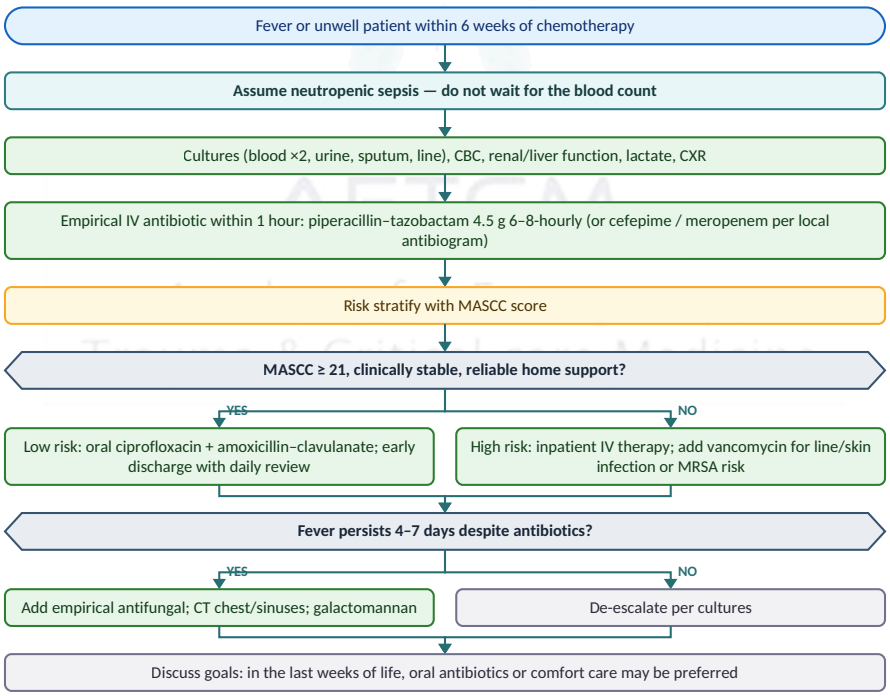
DEFINITION Cytotoxic chemotherapy toxicity, while drug-specific in detail, falls into recognisable classes that a palliative medicine postgraduate should be able to anticipate, screen for and manage across almost any regimen.

Toxicity Class	Typical Culprits / Note
Myelosuppression	Almost all cytotoxic chemotherapy; causes febrile neutropenia, anaemia, bleeding – the commonest dose-limiting and life-threatening toxicity class
Mucositis	Rapidly dividing mucosal cells affected by many cytotoxics; impairs oral intake and is a portal for infection
Cardiotoxicity	Anthracyclines (dose-dependent, often irreversible), trastuzumab (usually reversible) – requires baseline and serial cardiac monitoring
Nephrotoxicity	Platinum agents, high-dose methotrexate – requires hydration and monitoring protocols
Neurotoxicity	Vinca alkaloids (peripheral), platinum agents (peripheral), ifosfamide (encephalopathy)
Pulmonary toxicity	Bleomycin (fibrosis), some targeted/immunotherapies (pneumonitis)
Tumour lysis syndrome	Rapid cell death in highly chemosensitive, bulky tumours (Burkitt lymphoma, ALL, bulky lymphoma) releasing potassium, phosphate, uric acid

★ CLINICAL PEARLS & EXAM FOCUS

► Febrile neutropenia is a medical emergency regardless of which specific chemotherapy regimen caused it – empirical broad-spectrum antibiotics should never be delayed pending culture results.

ALGORITHM · Febrile Neutropenia Algo 15 — full page ›



2.1.11 Treatment-Related Complications – Radiotherapy, Surgery, Targeted & Immunotherapy

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Classify the complications of radiotherapy by timing, with examples. [Viva]

Q Discuss immune-related adverse events of checkpoint inhibitor immunotherapy. [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Anticipate and manage non-chemotherapy treatment-related complications across modalities (CD2.1, CD4.1, PD3)

DEFINITION Radiotherapy, surgery and the newer targeted/immunotherapy drug classes each carry distinct complication profiles that increasingly dominate the modern oncology complication landscape alongside classical chemotherapy toxicity.

Timing	Typical Complications
Acute (during/soon after treatment)	Mucositis/dermatitis within the treatment field, fatigue, nausea (site-dependent)
Late/chronic (months to years later)	Fibrosis, strictures (oesophageal, rectal, vaginal), radiation pneumonitis/fibrosis, secondary malignancy, hypopituitarism (cranial fields), cognitive decline (brain fields)

Surgical Complications

Timing	Typical Complications
Immediate	Bleeding, anaesthetic complications, wound infection
Early postoperative	Anastomotic leak, fistula formation, delayed wound healing
Late	Chronic pain, lymphoedema, functional/organ-specific deficits (stoma, continence, sexual function)

Targeted Therapy & Immunotherapy

Drug Class	Typical Complications
Anti-VEGF agents (bevacizumab and similar)	Hypertension, bleeding, impaired wound healing, GI perforation
Checkpoint inhibitor immunotherapy	Immune-related adverse events – colitis, pneumonitis, hepatitis, endocrinopathy (thyroiditis, hypophysitis, adrenal insufficiency), can affect any organ
Tyrosine kinase inhibitors (general class)	Hand-foot skin reaction, hypertension, diarrhoea, hepatotoxicity – specific profile varies by agent

★ CLINICAL PEARLS & EXAM FOCUS

▸ Immune-related adverse events can occur months after stopping checkpoint inhibitor therapy and can affect any organ system – maintain a high index of suspicion in any recently or previously immunotherapy-treated patient with a new symptom.

▸ High-dose corticosteroids are the mainstay of moderate-severe immune-related adverse event management, generally without reducing the anti-tumour benefit already achieved.

GUIDELINES & RECENT ADVANCES

- ASCO/ESMO Clinical Practice Guidelines: Management of Immune-Related Adverse Events (current version)

M1 Oral Cavity Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the complications of advanced oral cavity carcinoma and their palliative management. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Describe the palliative care needs and manage complications of head and neck malignancy (CD4.2, CD4.3)

DEFINITION Squamous cell carcinoma arising from the buccal mucosa, tongue, floor of mouth, alveolus or hard palate; strongly associated with tobacco chewing/smoking and alcohol, making it one of the commonest cancers in the Indian subcontinent.

Stage / Setting	Principal Treatment Approach
Early (T1-T2, N0)	Wide local excision or partial glossectomy/mandibulectomy ± neck dissection; may be followed by radiotherapy if adverse features
Locally advanced (T3-T4 or N+)	Surgery (composite resection, free-flap reconstruction) followed by adjuvant radiotherapy ± concurrent chemotherapy for high-risk features (positive margins, extracapsular nodal spread)
Metastatic/ unresectable	Palliative chemotherapy (platinum-based) ± cetuximab, or palliative radiotherapy for local control and bleeding/pain

COMPLICATIONS

Disease-related:

- Local invasion into mandible/maxilla causing pain, trismus and pathological fracture
- Dysphagia and aspiration from tongue/floor-of-mouth involvement
- Cervical lymph node metastasis with skin fungation
- Carotid artery erosion causing catastrophic haemorrhage in advanced/recurrent disease

Treatment-related:

- Mucositis and xerostomia from radiotherapy, severely impairing oral intake
- Osteoradionecrosis of the mandible
- Trismus from fibrosis of masticatory muscles post-radiotherapy
- Feeding-tube dependence (PEG) from surgical/radiation-related dysphagia

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation, high-risk adjuvant setting	100 mg/m² IV every 3 weeks for 3 cycles (or 40 mg/m² weekly)	Nephrotoxicity, ototoxicity, severe emesis; hydrate well and monitor renal function
Cetuximab	EGFR-targeted therapy, metastatic/ recurrent or with radiotherapy	400 mg/m² loading, then 250 mg/m² weekly IV	Acneiform rash, infusion reactions, hypomagnesaemia

★ CLINICAL PEARLS & EXAM FOCUS

▸ Anticipate and pre-emptively manage mucositis (oral care protocols, topical analgesia) before it becomes treatment-limiting.

▸ Pre-treatment dental assessment and extraction of unsalvageable teeth reduces osteoradionecrosis risk.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Head and Neck Cancers (current version)

M2 Oropharyngeal Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What are the complications of chemoradiation in oropharyngeal carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Describe the palliative management of treatment-related toxicity in head and neck cancer (CD4.2, PD4)

DEFINITION Squamous cell carcinoma of the tonsil, base of tongue or soft palate; increasingly driven by HPV (particularly type 16) in non-smokers, which carries a better prognosis than tobacco-related disease.

Stage / Setting	Principal Treatment Approach
Early, HPV-related	Transoral robotic surgery or definitive radiotherapy alone, given favourable prognosis
Locally advanced	Concurrent chemoradiation (cisplatin-based) as the mainstay, de-escalation trials ongoing for HPV-positive disease
Metastatic/ recurrent	Platinum-based chemotherapy with pembrolizumab or cetuximab; immunotherapy increasingly first-line

COMPLICATIONS

Disease-related:

- Dysphagia and odynophagia from base-of-tongue/tonsillar bulk
- Referred otalgia
- Cervical lymphadenopathy, sometimes the presenting sign (cystic neck node)
- Airway obstruction in bulky disease

Treatment-related:

- Severe radiation-induced mucositis and xerostomia
- Long-term dysphagia requiring swallowing rehabilitation
- Hypothyroidism from radiation to the neck
- Immune-related pneumonitis/colitis with checkpoint inhibitors

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation	100 mg/m² IV every 3 weeks for 3 cycles	Nephrotoxicity, ototoxicity, myelosuppression
Pembrolizumab	Recurrent/metastatic, PD-L1 expressing	200 mg IV every 3 weeks	Immune-related adverse events – pneumonitis, colitis, endocrinopathy

★ CLINICAL PEARLS & EXAM FOCUS

► HPV-related oropharyngeal cancer has a markedly better prognosis – counsel accordingly to avoid undue distress.

► Swallowing rehabilitation should begin proactively during chemoradiation, not only after dysphagia develops.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Head and Neck Cancers (current version)



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M3 Nasopharyngeal Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the clinical presentation and complications of nasopharyngeal carcinoma. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Relate anatomical site to the pattern of local complications in head and neck cancer (CD4.2, PD3)

DEFINITION Undifferentiated/poorly differentiated carcinoma strongly associated with Epstein-Barr virus infection, with a distinct geographic predilection (endemic in parts of East/Southeast Asia); presents with neck mass, nasal obstruction, epistaxis, or cranial nerve palsies from skull-base extension.

Stage / Setting	Principal Treatment Approach
Early (Stage I)	Radiotherapy alone to nasopharynx and neck
Locally advanced (Stage II-IVA)	Concurrent chemoradiation (cisplatin) followed by adjuvant chemotherapy (cisplatin-5FU or gemcitabine-based)
Metastatic	Gemcitabine-cisplatin combination chemotherapy; EBV DNA used to monitor response

COMPLICATIONS

Disease-related:

- Skull-base erosion with multiple cranial nerve palsies (III-VI, IX-XII)
- Nasal obstruction and recurrent epistaxis
- Cervical lymphadenopathy, often bilateral and the presenting complaint
- Distant metastasis to bone, liver and lung

Treatment-related:

- Severe xerostomia and dental caries from radiotherapy to the nasopharynx
- Radiation-induced temporal lobe necrosis (late complication)
- Hypopituitarism from skull-base radiation
- Trismus and hearing loss (radiation-induced otitis media)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation and adjuvant chemotherapy	100 mg/m² every 3 weeks (concurrent) or 80 mg/m² adjuvant	Nephrotoxicity, ototoxicity, nausea
Gemcitabine	Metastatic disease, combination with cisplatin	1000 mg/m² IV days 1 and 8 of a 21-day cycle	Myelosuppression, transaminitis

★ CLINICAL PEARLS & EXAM FOCUS

► Cranial nerve palsies at presentation should prompt urgent skull-base imaging rather than being attributed to a peripheral cause.

► EBV DNA titre is a useful, non-invasive marker of treatment response and recurrence.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Head and Neck Cancers (current version)

M4 Laryngeal Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss organ-preservation strategies and their complications in laryngeal carcinoma. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage airway and voice-related complications of laryngeal cancer in a palliative care setting (CD4.2, PD8)

DEFINITION Squamous cell carcinoma of the glottis, supraglottis or subglottis, closely linked to tobacco and alcohol use; glottic tumours present early with hoarseness, while supraglottic tumours present later with a neck mass or dysphagia.

Stage / Setting	Principal Treatment Approach
Early glottic (T1-T2)	Radiotherapy or trans-oral laser surgery, both with excellent voice-preservation and cure rates
Locally advanced	Organ-preservation concurrent chemoradiation, or total laryngectomy with adjuvant (chemo)radiotherapy if non-preservable
Metastatic/ recurrent	Palliative chemotherapy ± immunotherapy; palliative radiotherapy for bleeding/airway symptoms

COMPLICATIONS

Disease-related:

- Airway obstruction requiring emergency tracheostomy
- Aspiration pneumonia from impaired laryngeal closure
- Direct extension to thyroid cartilage and strap muscles
- Nodal and distant metastasis (lung most common)

Treatment-related:

- Permanent tracheostomy/stoma care needs after total laryngectomy
- Loss of natural voice, requiring speech rehabilitation (oesophageal speech, electrolarynx, tracheo-oesophageal puncture)
- Pharyngocutaneous fistula after laryngectomy
- Radiation-induced laryngeal oedema and chondronecrosis

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation (organ preservation)	100 mg/m² every 3 weeks for 3 cycles	Nephrotoxicity, ototoxicity
Nivolumab	Platinum-refractory recurrent/metastatic disease	240 mg IV every 2 weeks	Immune-related adverse events

★ CLINICAL PEARLS & EXAM FOCUS

► Hoarseness persisting beyond 3 weeks in a smoker warrants laryngoscopy – a frequently tested red flag.

► Stoma and tracheostomy care education for family is essential before discharge after laryngectomy.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Head and Neck Cancers (current version)

M5 Hypopharyngeal Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why does hypopharyngeal carcinoma carry a worse prognosis than glottic carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Integrate early palliative care in poor-prognosis head and neck malignancy (CD4.1, AD2)

DEFINITION Squamous cell carcinoma of the pyriform sinus, postcricoid region or posterior pharyngeal wall; often presents late (painless neck mass, dysphagia) due to a silent anatomical location, carrying the worst prognosis among head and neck subsites.

Stage / Setting	Principal Treatment Approach
Early (rare at diagnosis)	Partial pharyngolaryngectomy or radiotherapy with organ preservation intent
Locally advanced (majority)	Concurrent chemoradiation or total laryngopharyngectomy with reconstruction, followed by adjuvant therapy
Metastatic	Palliative chemotherapy and best supportive care; high symptom burden from dysphagia

COMPLICATIONS

Disease-related:

- Early submucosal spread with a high rate of nodal and distant metastasis at presentation
- Severe dysphagia and aspiration
- Oesophageal stricture from submucosal extension
- Direct invasion of larynx causing airway compromise

Treatment-related:

- High rate of pharyngocutaneous fistula after surgery
- Permanent dysphagia requiring long-term feeding-tube support
- Radiation fibrosis worsening swallowing function
- Second primary tumours of the upper aerodigestive tract (field cancerisation)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation	100 mg/m² every 3 weeks for 3 cycles	Nephrotoxicity, mucositis compounding dysphagia
5-Fluorouracil	Combination palliative chemotherapy	1000 mg/m²/day continuous infusion days 1-4	Mucositis, diarrhoea, hand-foot syndrome

★ CLINICAL PEARLS & EXAM FOCUS

► Given late presentation and poor prognosis, early integration of palliative care alongside oncologic treatment is particularly important here.

► Nutritional support planning (PEG) should be proactive, as dysphagia is near-universal.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Head and Neck Cancers (current version)

M6 Salivary Gland Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is facial nerve palsy a worrying sign in a parotid mass? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise distinct biological behaviour across head and neck cancer histologies (CD4.2)

DEFINITION A heterogeneous group of malignancies (mucoepidermoid carcinoma, adenoid cystic carcinoma, acinic cell carcinoma) most commonly arising in the parotid gland; adenoid cystic carcinoma is notorious for perineural spread and very late (even decade-delayed) distant recurrence, typically to lung.

Stage / Setting	Principal Treatment Approach
Low-grade, localised	Surgical excision (superficial/total parotidectomy) with facial nerve preservation where feasible
High-grade or perineural invasion	Surgery followed by adjuvant radiotherapy, particularly for adenoid cystic histology
Metastatic (especially adenoid cystic, lung-only)	Often observed given indolent growth; systemic therapy reserved for symptomatic/ rapidly progressive disease

COMPLICATIONS

Disease-related:

- Facial nerve palsy from tumour infiltration (suggests malignancy over benign parotid tumour)
- Perineural spread along cranial nerves (adenoid cystic carcinoma) causing pain and numbness
- Indolent but eventual pulmonary metastasis, sometimes stable for years
- Local recurrence due to microscopic perineural extension beyond surgical margins

Treatment-related:

- Permanent facial nerve palsy after radical parotidectomy, needing eye protection and physiotherapy
- Frey's syndrome (gustatory sweating) after parotidectomy
- Xerostomia from adjuvant radiotherapy
- First-bite syndrome after deep lobe parotid surgery

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Carboplatin + Paclitaxel	Symptomatic metastatic disease (select histologies)	Carboplatin AUC 5 + paclitaxel 175 mg/m² every 3 weeks	Myelosuppression, peripheral neuropathy
Lenvatinib	Advanced/progressive disease, selected centres	20 mg orally once daily	Hypertension, proteinuria, fatigue

★ CLINICAL PEARLS & EXAM FOCUS

► New facial nerve weakness with a parotid mass should always raise suspicion of malignancy.

► Lung-only metastatic adenoid cystic carcinoma may remain stable for years – active surveillance before systemic therapy is often appropriate.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Head and Neck Cancers (current version)

M7 Papillary Thyroid Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the complications of total thyroidectomy. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage postoperative complications of endocrine malignancy surgery (CD4.2, PD8)

DEFINITION The commonest thyroid malignancy, typically indolent with an excellent prognosis, arising from follicular cells; often presents as a painless thyroid nodule, with spread preferentially to cervical lymph nodes.

Stage / Setting	Principal Treatment Approach
Low-risk, localised	Lobectomy or total thyroidectomy depending on size/risk factors; active surveillance an option for very low-risk microcarcinoma
Intermediate/high-risk	Total thyroidectomy with central/lateral neck dissection, followed by radioactive iodine (I-131) ablation
Metastatic/radioiodine-refractory	Tyrosine kinase inhibitors (lenvatinib, sorafenib) for progressive disease

COMPLICATIONS

Disease-related:

- Cervical lymph node metastasis, often the presenting feature
- Recurrent laryngeal nerve invasion causing vocal cord palsy
- Tracheal/oesophageal invasion in locally advanced disease
- Distant metastasis to lung and bone (uncommon but possible with long disease course)

Treatment-related:

- Permanent hypoparathyroidism (hypocalcaemia) after total thyroidectomy
- Recurrent laryngeal nerve injury during surgery causing hoarseness
- Lifelong levothyroxine dependence and risk of iatrogenic thyrotoxicosis if over-replaced
- Sialadenitis from radioactive iodine therapy

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Levothyroxine	TSH-suppressive therapy post-thyroidectomy	Titrated to suppress TSH, typically 1.6-2.0 µg/kg/day	Over-suppression risks atrial fibrillation and osteoporosis, especially in elderly
Lenvatinib	Radioiodine-refractory progressive metastatic disease	24 mg orally once daily	Hypertension, proteinuria, GI perforation risk, fatigue

★ CLINICAL PEARLS & EXAM FOCUS

► Post-thyroidectomy hypocalcaemia (circumoral tingling, Chvostek sign) must be checked for and treated promptly.

► Prognosis is excellent – reassurance and avoiding over-treatment anxiety is an important palliative/communication skill even though 'cancer' is in the diagnosis.

GUIDELINES & RECENT ADVANCES

- American Thyroid Association Management Guidelines for Thyroid Nodules and DTC

M8 Follicular Thyroid Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How does the spread pattern of follicular carcinoma differ from papillary carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Relate tumour biology to metastatic pattern and complications (CD4.2)

DEFINITION The second commonest differentiated thyroid cancer, distinguished from papillary carcinoma by capsular/ vascular invasion rather than nuclear features; spreads haematogenously (bone, lung) rather than via lymphatics.

Stage / Setting	Principal Treatment Approach
Minimally invasive	Lobectomy may suffice if capsular invasion only, no vascular invasion
Widely invasive	Total thyroidectomy followed by radioactive iodine ablation
Metastatic (bone/ lung)	Radioiodine therapy if iodine-avid; tyrosine kinase inhibitors if refractory; bisphosphonates/denosumab and radiotherapy for bone metastases

COMPLICATIONS

Disease-related:

- Haematogenous spread to bone causing pathological fracture
- Pulmonary metastases, sometimes miliary and iodine-avid
- Vascular invasion increasing risk of distant spread at diagnosis
- Vertebral metastasis with risk of cord compression

Treatment-related:

- Hypothyroidism/hypoparathyroidism after total thyroidectomy
- Cumulative radioiodine toxicity – marrow suppression, rarely secondary leukaemia
- Bone pain and fracture risk from skeletal metastases
- Osteonecrosis of jaw from bone-targeted agents

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Radioactive Iodine (I-131)	Ablation/treatment of iodine-avid metastatic disease	Activity individualised (typically 100-200 mCi for metastatic disease)	Sialadenitis, bone marrow suppression at high cumulative doses, secondary malignancy risk
Zoledronic acid	Bone metastases	4 mg IV every 3-4 weeks	Osteonecrosis of jaw, renal function monitoring required

★ CLINICAL PEARLS & EXAM FOCUS

► Unlike papillary carcinoma, nodal spread is uncommon – distant (bone/lung) spread should be actively looked for.

► Bone pain in a patient with known follicular carcinoma should prompt imaging even years after initial treatment.

GUIDELINES & RECENT ADVANCES

- American Thyroid Association Management Guidelines for Thyroid Nodules and DTC

M9 Medullary Thyroid Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must phaeochromocytoma be excluded before surgery for medullary thyroid carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise syndromic associations relevant to palliative oncology (CD4.2, CD9)

DEFINITION Arises from parafollicular (C) cells and secretes calcitonin, useful as a tumour marker; occurs sporadically or as part of MEN 2A/2B syndromes (RET proto-oncogene mutations), making genetic counselling and screening of relatives important.

Stage / Setting	Principal Treatment Approach
Localised	Total thyroidectomy with prophylactic central neck dissection (and lateral dissection if nodes involved)
Locally advanced/ nodal	Total thyroidectomy with extensive neck dissection; radiotherapy for residual/unresectable disease
Metastatic/RET-altered	Selective RET inhibitors (selpercatinib, pralsetinib) for RET-mutant disease; vandetanib/ cabozantinib otherwise

COMPLICATIONS

Disease-related:

- Secretory diarrhoea from excess calcitonin/other peptides, a distinctive and distressing symptom
- Associated phaeochromocytoma (MEN 2) causing hypertensive crises if undiagnosed before surgery
- Cervical and mediastinal nodal metastasis
- Liver and bone metastases in advanced disease

Treatment-related:

- Permanent hypoparathyroidism after extensive neck dissection
- QT prolongation and arrhythmia risk with RET/multikinase inhibitors
- Hepatotoxicity requiring regular liver function monitoring on targeted therapy
- Diarrhoea from calcitonin excess, sometimes worsened by targeted therapy itself

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Selpercatinib	RET-mutant metastatic disease	160 mg orally twice daily	Hepatotoxicity, hypertension, QT prolongation
Vandetanib	Progressive symptomatic metastatic disease	300 mg orally once daily	QT prolongation, diarrhoea, skin toxicity – requires ECG monitoring

★ CLINICAL PEARLS & EXAM FOCUS

► Always screen for phaeochromocytoma before thyroid surgery in suspected MEN 2 to avoid an intraoperative hypertensive crisis.

► Calcitonin and CEA levels are useful markers for monitoring disease burden and treatment response.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Thyroid Carcinoma (current version)

M10 Anaplastic Thyroid Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the approach to goals-of-care discussion in anaplastic thyroid carcinoma. [Paper 3, ethics]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply ethical principles and communication skills in rapidly fatal malignancy (AD2, PD1, PD2)

DEFINITION A rare but extremely aggressive, undifferentiated thyroid malignancy typically presenting in older adults with a rapidly enlarging neck mass, often from dedifferentiation of a pre-existing differentiated thyroid cancer; median survival is measured in months.

Stage / Setting	Principal Treatment Approach
Localised (rare)	Aggressive multimodality therapy: surgery, radiotherapy and chemotherapy if resectable and performance status allows
Locally advanced/airway-threatening	Urgent tracheostomy for airway protection; palliative radiotherapy; BRAF/MEK inhibitors if BRAF V600E mutated
Metastatic/poor performance status	Best supportive/palliative care is often the most appropriate option given dismal prognosis

COMPLICATIONS

Disease-related:

- Rapid airway obstruction requiring emergency tracheostomy
- Direct invasion of trachea, oesophagus and great vessels
- Rapid, early distant metastasis (lung, bone, brain)
- Superior vena cava obstruction from mediastinal extension

Treatment-related:

- Tracheostomy-related complications (bleeding, tube displacement) in a friable, invaded airway
- Radiation-induced skin breakdown over a bulky, fungating neck mass
- Rapid clinical deterioration limiting ability to tolerate systemic therapy
- Significant psychological distress from an extremely rapid disease course

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dabrafenib + Trametinib	BRAF V600E-mutated disease	Dabrafenib 150 mg twice daily + trametinib 2 mg once daily	Fever, rash, cardiomyopathy – requires close monitoring
Dexamethasone	Peritumoural oedema/ airway symptoms, adjunct	4-8 mg twice daily, tapered as tolerated	Hyperglycaemia, proximal myopathy with prolonged use

★ CLINICAL PEARLS & EXAM FOCUS

▸ Early, explicit goals-of-care discussion is essential given the uniformly poor prognosis – this is a classic exam scenario for discussing realistic expectations.

▸ BRAF V600E testing should be performed urgently given the dramatic (if often transient) responses possible with targeted therapy.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Thyroid Carcinoma (current version)

M11 Non-Small Cell Lung Carcinoma – Adenocarcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role of molecular testing in management of metastatic lung adenocarcinoma. [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply biomarker-directed systemic therapy principles to oncology practice (CD4.1, CD2.1)

DEFINITION The commonest subtype of lung cancer overall, arising peripherally, increasingly seen in never-smokers (especially with EGFR/ALK/ROS1 alterations); presents with cough, haemoptysis, weight loss or incidental finding.

Stage / Setting	Principal Treatment Approach
Early (Stage I-II)	Surgical resection (lobectomy) ± adjuvant chemotherapy/targeted therapy or immunotherapy based on biomarker status
Locally advanced (Stage III)	Concurrent chemoradiation followed by consolidation durvalumab (immunotherapy)
Metastatic (Stage IV)	Biomarker-driven therapy – targeted agents (EGFR/ALK/ROS1 inhibitors) if actionable mutation present, otherwise platinum doublet chemotherapy ± immunotherapy

COMPLICATIONS

Disease-related:

- Malignant pleural effusion causing breathlessness
- Brain metastases, common even at presentation with adenocarcinoma
- Bone metastases with pain and pathological fracture risk
- Superior vena cava obstruction from mediastinal nodal disease

Treatment-related:

- Checkpoint-inhibitor pneumonitis, which can mimic disease progression on imaging
- Osimertinib-related interstitial lung disease
- Radiation pneumonitis after thoracic radiotherapy
- Chemotherapy-induced myelosuppression and febrile neutropenia

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Osimertinib	EGFR-mutant (exon 19 del/ L858R) metastatic disease	80 mg orally once daily	Interstitial lung disease, QT prolongation, diarrhoea
Pembrolizumab	PD-L1 high, no actionable mutation	200 mg IV every 3 weeks	Immune-related pneumonitis, colitis, endocrinopathy
Carboplatin + Pemetrexed	Non-squamous, no actionable mutation/low PD-L1	Carboplatin AUC 5 + pemetrexed 500 mg/m² every 3 weeks	Myelosuppression, renal impairment with pemetrexed

★ CLINICAL PEARLS & EXAM FOCUS

► Molecular testing (EGFR, ALK, ROS1, PD-L1) should be sent upfront in advanced non-squamous NSCLC – it changes first-line treatment entirely.

► New breathlessness in a known NSCLC patient needs effusion, pneumonitis and progression all considered, not just 'disease progression'.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Non-Small Cell Lung Cancer (current version)

M12 Non-Small Cell Lung Carcinoma – Squamous Cell

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What paraneoplastic syndrome is classically associated with squamous cell lung carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise paraneoplastic syndromes and their emergency management (CD4.1, PD4)

DEFINITION Centrally located NSCLC subtype, strongly linked to smoking, arising from bronchial epithelium; can cavitate and is associated with hypercalcaemia from PTHrP secretion (a paraneoplastic syndrome).

Stage / Setting	Principal Treatment Approach
Early (Stage I-II)	Surgical resection ± adjuvant chemotherapy or immunotherapy
Locally advanced (Stage III)	Concurrent chemoradiation followed by consolidation immunotherapy
Metastatic (Stage IV)	Platinum doublet chemotherapy (avoiding pemetrexed, which is ineffective in squamous histology) ± immunotherapy

COMPLICATIONS

Disease-related:

- Central airway obstruction with post-obstructive pneumonia
- Cavitation with secondary infection or haemoptysis
- Paraneoplastic hypercalcaemia from PTHrP secretion
- Direct invasion of chest wall and brachial plexus (superior sulcus/Pancoast variant)

Treatment-related:

- Massive haemoptysis from centrally invasive or cavitating tumour
- Radiation-induced oesophagitis from thoracic radiotherapy fields
- Bronchopleural fistula after surgery or necrosis
- Chemotherapy-related peripheral neuropathy limiting function

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Carboplatin + Paclitaxel + Pembrolizumab	First-line metastatic squamous NSCLC	Carboplatin AUC 6 + paclitaxel 200 mg/m² + pembrolizumab 200 mg, every 3 weeks	Myelosuppression, peripheral neuropathy, immune-related toxicity
Nintedanib	Second-line, select cases with adenocarcinoma component	200 mg orally twice daily	Diarrhoea, hepatotoxicity, bleeding risk

★ CLINICAL PEARLS & EXAM FOCUS

► Pemetrexed should not be used in squamous histology – a frequently tested pharmacology point.

► Hypercalcaemia in a smoker with a lung mass strongly suggests squamous histology and PTHrP-mediated paraneoplastic syndrome.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Non-Small Cell Lung Cancer (current version)

M13 Small Cell Lung Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q List the paraneoplastic syndromes associated with small cell lung carcinoma. [Paper 2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Describe paraneoplastic endocrine and neurological syndromes in palliative oncology (CD4.1, CD9.1)

DEFINITION A highly aggressive neuroendocrine lung cancer, almost exclusively in smokers, characterised by rapid growth, early widespread metastasis, and a high rate of paraneoplastic syndromes (SIADH, Lambert-Eaton myasthenic syndrome, Cushing's from ectopic ACTH).

Stage / Setting	Principal Treatment Approach
Limited stage (confined to one hemithorax)	Concurrent chemoradiation followed by prophylactic cranial irradiation if good response
Extensive stage (metastatic)	Platinum-etoposide chemotherapy combined with immunotherapy (atezolizumab/ durvalumab)
Relapsed/refractory	Topotecan or lurbinectedin; generally poor response durability

COMPLICATIONS

Disease-related:

- Early, widespread metastasis to liver, bone, brain and adrenal glands
- SIADH causing hyponatraemia
- Ectopic ACTH secretion causing Cushing syndrome and severe hypokalaemia
- Lambert-Eaton myasthenic syndrome (proximal weakness improving with repeated effort)

Treatment-related:

- Severe, often prolonged myelosuppression from platinum-etoposide chemotherapy
- Tumour lysis syndrome given rapid chemosensitivity
- Radiation-induced oesophagitis and pneumonitis
- Neurocognitive effects of prophylactic cranial irradiation

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin + Etoposide	First-line limited and extensive stage	Cisplatin 75-80 mg/m² day 1 + etoposide 100 mg/m² days 1-3, every 3 weeks	Myelosuppression, nephrotoxicity, severe emesis
Atezolizumab	Added to chemotherapy, extensive stage	1200 mg IV every 3 weeks with chemotherapy, then maintenance	Immune-related adverse events

★ CLINICAL PEARLS & EXAM FOCUS

▸ SIADH-related hyponatraemia in a smoker with a lung mass should immediately raise suspicion of small cell carcinoma.

▸ Given rapid chemosensitivity, anticipate and pre-emptively manage tumour lysis syndrome at treatment initiation in bulky disease.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Small Cell Lung Cancer (current version)

M14 Malignant Pleural Mesothelioma

Practice Qs ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the pain management challenges in malignant pleural mesothelioma. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage refractory chest wall pain using multimodal and interventional analgesia (PD3, CD3.1)

DEFINITION An aggressive tumour of the pleural mesothelium strongly linked to asbestos exposure, with a long latency (20-40 years) between exposure and diagnosis; presents with breathlessness and chest pain from pleural thickening/effusion.

Stage / Setting	Principal Treatment Approach
Localised, resectable (rare)	Extrapleural pneumonectomy or pleurectomy/decortication in highly selected, fit patients, combined with chemotherapy/radiotherapy
Locally advanced/ unresectable	Systemic chemotherapy (pemetrexed-platinum) ± bevacizumab
Metastatic/relapsed	Immunotherapy combination (nivolumab-ipilimumab) or further chemotherapy

COMPLICATIONS

Disease-related:

- Recurrent, often loculated pleural effusion refractory to simple drainage
- Direct chest wall invasion causing severe, often neuropathic pain
- Diaphragmatic and pericardial invasion
- Tumour seeding along prior biopsy/drain tracts (requiring prophylactic irradiation historically)

Treatment-related:

- Pleurodesis failure due to a trapped, non-expansile lung
- Immune-related colitis/pneumonitis with checkpoint inhibitor combinations
- Renal impairment from cisplatin limiting further treatment
- Severe chest wall pain often poorly responsive to opioids alone, needing multimodal/interventional approaches

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Pemetrexed + Cisplatin	First-line standard of care	Pemetrexed 500 mg/m² + cisplatin 75 mg/m² every 3 weeks	Myelosuppression, renal impairment; requires folate/B12 supplementation
Nivolumab + Ipilimumab	First-line alternative, unresectable disease	Nivolumab 3 mg/kg every 2 weeks + ipilimumab 1 mg/kg every 6 weeks	Immune-related adverse events, can be severe/multi-organ

★ CLINICAL PEARLS & EXAM FOCUS

▸ Mesothelioma pain is often severe and neuropathic (chest wall invasion) – adjuvant analgesics and early interventional referral should not be delayed.

▸ An occupational/asbestos exposure history should always be taken, with medico-legal/compensation implications discussed where relevant.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Malignant Pleural Mesothelioma (current version)

M15 Thymoma / Thymic Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must myasthenia gravis be screened for before thymectomy? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise paraneoplastic neuromuscular syndromes relevant to perioperative risk (CD4.1, CD9.1)

DEFINITION Tumours of the anterior mediastinum arising from thymic epithelium; thymoma is often indolent and associated with myasthenia gravis, while thymic carcinoma is more aggressive with a higher metastatic potential.

Stage / Setting	Principal Treatment Approach
Localised, encapsulated (Masaoka I-II)	Complete surgical resection via median sternotomy; adjuvant radiotherapy if positive margins
Locally invasive (Masaoka III-IVA)	Surgery combined with (neo)adjuvant chemotherapy and radiotherapy
Metastatic/unresectable	Platinum-based chemotherapy (CAP regimen); sunitinib or other targeted agents for thymic carcinoma

COMPLICATIONS

Disease-related:

- Myasthenia gravis with risk of myasthenic crisis and respiratory failure
- Superior vena cava obstruction from mediastinal compression
- Pleural and pericardial seeding (drop metastases)
- Pure red cell aplasia and hypogammaglobulinaemia (paraneoplastic associations)

Treatment-related:

- Perioperative myasthenic crisis requiring ventilatory support
- Cardiotoxicity from anthracycline-based chemotherapy
- Radiation pneumonitis/fibrosis from mediastinal radiotherapy
- Post-thymectomy exacerbation of myasthenia gravis

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cyclophosphamide + Doxorubicin + Cisplatin (CAP)	Advanced/ unresectable thymoma or thymic carcinoma	Per institutional protocol, every 3 weeks	Cardiotoxicity (doxorubicin), myelosuppression, nephrotoxicity
Pyridostigmine	Associated myasthenia gravis symptom control	30-60 mg orally 3-4 times daily, titrated	Cholinergic excess – cramps, diarrhoea, salivation

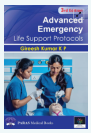
★ CLINICAL PEARLS & EXAM FOCUS

► Always screen for myasthenia gravis symptoms (ptosis, fatigable weakness) in a patient with an anterior mediastinal mass before surgery/anaesthesia.

► Anaesthetic and perioperative planning must anticipate myasthenic crisis risk.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Thymomas and Thymic Carcinomas (current version)



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M16 Bronchopulmonary Carcinoid Tumour

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What precaution is taken before surgery in a functioning carcinoid tumour? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Anticipate and prevent hormone-mediated perioperative crises in neuroendocrine tumours (CD4.1, PD3)

DEFINITION A low- to intermediate-grade neuroendocrine tumour of the bronchus, typically presenting in younger patients with haemoptysis, recurrent pneumonia (from bronchial obstruction) or incidentally; carcinoid syndrome is uncommon unless hepatic metastases are present.

Stage / Setting	Principal Treatment Approach
Typical carcinoid, localised	Surgical resection (lobectomy, sleeve resection or bronchotomy for central lesions) is usually curative
Atypical carcinoid or nodal involvement	Anatomical resection with lymphadenectomy; adjuvant therapy considered for high-risk features
Metastatic (liver-dominant)	Somatostatin analogues for symptom/biochemical control; liver-directed therapy (embolisation) or peptide receptor radionuclide therapy

COMPLICATIONS

Disease-related:

- Bronchial obstruction causing recurrent post-obstructive pneumonia
- Carcinoid syndrome (flushing, diarrhoea, bronchospasm) if liver metastases present
- Carcinoid heart disease (tricuspid/pulmonary valve fibrosis) in long-standing syndrome
- Haemoptysis from a vascular, centrally located tumour

Treatment-related:

- Carcinoid crisis (severe hypotension, bronchospasm) precipitated by anaesthesia or tumour manipulation without pre-treatment octreotide
- Gallstone formation with long-term somatostatin analogue use
- Injection-site reactions and local complications of depot therapy
- Rarely, embolisation-related post-embolisation syndrome (pain, fever) after liver-directed therapy

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Octreotide LAR	Symptomatic carcinoid syndrome/ biochemical control	20-30 mg IM every 4 weeks	Gallstones, injection-site reactions, mild hyperglycaemia
Lanreotide	Alternative somatostatin analogue, antiproliferative use	120 mg deep SC every 4 weeks	Similar to octreotide; GI discomfort

★ CLINICAL PEARLS & EXAM FOCUS

► Pre-operative octreotide cover is essential before surgery/biopsy of a functioning carcinoid to prevent an intraoperative carcinoid crisis.

► Unlike most lung cancers, bronchial carcinoid has an excellent prognosis after complete resection – important for accurate patient counselling.

GUIDELINES & RECENT ADVANCES

- NANETS/ENETS Consensus Guidelines: Bronchopulmonary Neuroendocrine Tumours

M17 Breast Carcinoma – Hormone Receptor-Positive

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the first-line management of hormone receptor-positive metastatic breast cancer. [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply endocrine and targeted therapy principles in breast cancer palliative care (CD4.1, CD2.1)

DEFINITION The commonest breast cancer subtype (ER/PR-positive, HER2-negative), typically slower-growing with late relapse risk persisting beyond 10 years; the mainstay of treatment is endocrine therapy rather than chemotherapy in most cases.

Stage / Setting	Principal Treatment Approach
Early stage	Breast-conserving surgery or mastectomy with sentinel node biopsy, followed by adjuvant endocrine therapy ± chemotherapy per recurrence-score risk
Locally advanced	Neoadjuvant chemotherapy or endocrine therapy followed by surgery and radiotherapy
Metastatic	Endocrine therapy combined with a CDK4/6 inhibitor as first-line in most patients; chemotherapy reserved for visceral crisis or endocrine-refractory disease

COMPLICATIONS

Disease-related:

- Bone metastases causing pathological fracture and hypercalcaemia
- Late relapse (sometimes 10-15 years after diagnosis), unique to this subtype
- Endometrial hyperplasia/carcinoma with long-term tamoxifen use
- Leptomeningeal disease in advanced, long-standing metastatic disease

Treatment-related:

- Venous thromboembolism on tamoxifen
- Neutropenia and febrile neutropenia risk with CDK4/6 inhibitors
- Osteoporosis/fracture risk from aromatase inhibitors (oestrogen deprivation)
- Hot flushes and sexual dysfunction significantly affecting quality of life

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Tamoxifen	Adjuvant/metastatic, pre- and peri-menopausal	20 mg orally once daily for 5-10 years	Endometrial cancer risk, venous thromboembolism, hot flushes
Letrozole + Palbociclib	First-line metastatic, postmenopausal	Letrozole 2.5 mg once daily + palbociclib 125 mg once daily (3 weeks on/1 week off)	Neutropenia (palbociclib), fatigue; monitor blood counts

★ CLINICAL PEARLS & EXAM FOCUS

▸ CDK4/6 inhibitor + endocrine therapy, not chemotherapy, is now standard first-line for most HR-positive metastatic disease – an important recent-advances point.

▸ Bone-targeted agents (bisphosphonate/denosumab) should be started early given the high incidence of bone-only metastatic disease.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Breast Cancer (current version)

M18 Breast Carcinoma – HER2-Positive

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What monitoring is required during trastuzumab therapy and why? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Monitor and manage targeted-therapy cardiotoxicity (CD4.1, PD9)

DEFINITION A biologically aggressive subtype driven by HER2/neu amplification, historically associated with poor prognosis before the advent of anti-HER2 targeted therapy, which has transformed outcomes.

Stage / Setting	Principal Treatment Approach
Early stage	Neoadjuvant chemotherapy with dual anti-HER2 blockade (trastuzumab + pertuzumab), surgery, then completion of targeted therapy for 1 year
Locally advanced	Neoadjuvant chemo-targeted therapy followed by surgery and radiotherapy; adjuvant T-DM1 if residual disease
Metastatic	Taxane + trastuzumab + pertuzumab first-line; trastuzumab deruxtecan on progression

COMPLICATIONS

Disease-related:

- Brain metastases, disproportionately common in HER2-positive disease even with good systemic control
- Cardiac dysfunction from the disease/treatment interaction
- Liver and bone metastases in advanced disease
- Leptomeningeal spread in heavily pretreated disease

Treatment-related:

- Trastuzumab-induced cardiotoxicity, usually reversible on discontinuation
- Interstitial lung disease with trastuzumab deruxtecan, a serious and potentially fatal toxicity
- Peripheral neuropathy from taxane-based chemotherapy
- Diarrhoea from dual anti-HER2 blockade

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Trastuzumab	All stages of HER2-positive disease	8 mg/kg loading then 6 mg/kg IV every 3 weeks	Cardiotoxicity (reversible LV dysfunction) – requires baseline and serial echocardiography
Trastuzumab Deruxtecan	Second-line+ metastatic disease	5.4 mg/kg IV every 3 weeks	Interstitial lung disease/pneumonitis – potentially fatal, requires vigilant monitoring

★ CLINICAL PEARLS & EXAM FOCUS

► Baseline and 3-monthly cardiac function (ECHO/MUGA) monitoring is mandatory throughout anti-HER2 therapy.

► Any new respiratory symptom on trastuzumab deruxtecan must trigger urgent evaluation for interstitial lung disease.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Breast Cancer (current version)

M19 Breast Carcinoma – Triple-Negative

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role of BRCA testing in triple-negative breast cancer management. [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Integrate genetic counselling considerations into oncology palliative practice (CD4.1, CD9)

DEFINITION A biologically heterogeneous, aggressive subtype lacking ER, PR and HER2 expression, more common in younger women and BRCA1 mutation carriers, with a higher early relapse rate and reliance on chemotherapy/immunotherapy rather than targeted endocrine or anti-HER2 agents.

Stage / Setting	Principal Treatment Approach
Early stage	Neoadjuvant chemotherapy (anthracycline-taxane ± carboplatin) with pembrolizumab if high-risk; surgery; adjuvant capecitabine if residual disease
Locally advanced	Neoadjuvant chemo-immunotherapy followed by surgery and radiotherapy
Metastatic	Chemotherapy ± immunotherapy (PD-L1 positive); sacituzumab govitecan or PARP inhibitor (if BRCA-mutated) on progression

COMPLICATIONS

Disease-related:

- Higher rate of early (within 2-3 years) visceral and brain relapse compared with other subtypes
- Rapid symptomatic progression once metastatic given aggressive biology
- BRCA-associated risk of contralateral breast and ovarian cancer
- Chemotherapy-refractory disease emerging relatively early in the metastatic course

Treatment-related:

- Severe neutropenia/febrile neutropenia with combination chemotherapy
- Immune-related colitis/hepatitis/pneumonitis with pembrolizumab
- Secondary myelodysplasia/leukaemia, a rare but serious late effect of PARP inhibitors
- Significant psychosocial distress given younger age at diagnosis and aggressive course

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Pembrolizumab	Neoadjuvant/adjuvant high-risk, or metastatic PD-L1 positive	200 mg IV every 3 weeks, with chemotherapy	Immune-related adverse events
Sacituzumab Govitecan	Second-line+ metastatic disease	10 mg/kg IV days 1 and 8 of a 21-day cycle	Neutropenia, diarrhoea – requires growth factor support and antidiarrhoeal readiness
Olaparib	BRCA-mutated, metastatic	300 mg orally twice	Myelosuppression, fatigue, rare
	or high-risk adjuvant	daily	secondary MDS/AML

★ CLINICAL PEARLS & EXAM FOCUS

▸ BRCA testing should be offered to all triple-negative breast cancer patients, particularly those diagnosed young – it has both therapeutic (PARP inhibitor) and family-screening implications.

▸ Given the aggressive trajectory, early, proactive goals-of-care conversations are particularly valuable in this subtype.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Breast Cancer (current version)

M20 Inflammatory Breast Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What clinical clue distinguishes inflammatory breast carcinoma from mastitis? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise atypical presentations of breast malignancy requiring urgent referral (CD4.1)

DEFINITION A rare, highly aggressive clinical-pathological entity presenting with rapid-onset breast erythema, oedema ('peau d'orange'), warmth and induration due to dermal lymphatic tumour emboli, often without a discrete palpable mass, and frequently misdiagnosed initially as mastitis.

Stage / Setting	Principal Treatment Approach
All stages (treated as locally advanced at minimum)	Neoadjuvant chemotherapy (± targeted/immunotherapy by receptor subtype) is mandatory before any local therapy
After neoadjuvant response	Modified radical mastectomy (breast-conserving surgery is contraindicated) with axillary dissection
Post-surgery	Adjuvant chest wall and regional nodal radiotherapy is routine given the high locoregional recurrence risk

COMPLICATIONS

Disease-related:

- Rapid lymphatic and haematogenous dissemination given dermal lymphatic invasion at diagnosis
- Skin ulceration and secondary infection of the inflamed breast
- Chest wall recurrence despite mastectomy, given the diffuse dermal involvement
- High rate of distant metastasis at diagnosis compared with other breast cancer presentations

Treatment-related:

- Wound healing complications after mastectomy in previously irradiated or infected skin
- Radiation-induced skin breakdown over already compromised chest wall skin
- Lymphoedema of the arm from axillary dissection and nodal radiotherapy
- Delay in diagnosis (mistaken for mastitis) worsening outcomes – an important teaching point

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Doxorubicin + Cyclophosphamide, then Paclitaxel	Standard neoadjuvant backbone	Per institutional dose-dense or standard protocol	Cardiotoxicity (anthracycline), myelosuppression, peripheral neuropathy (taxane)
Trastuzumab + Pertuzumab	If HER2-positive (added to chemotherapy backbone)	Per standard HER2-targeted dosing schedule	Cardiotoxicity – monitor LVEF

★ CLINICAL PEARLS & EXAM FOCUS

▸ Any 'mastitis' in a non-lactating woman that fails to resolve with antibiotics within 1-2 weeks warrants urgent biopsy to exclude inflammatory breast cancer.

▸ Breast-conserving surgery is contraindicated in inflammatory breast cancer – mastectomy is mandatory.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Breast Cancer (current version)

M21 Male Breast Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What hereditary association is important to screen for in male breast cancer? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise rare malignancy presentations and their psychosocial impact (CD4.1, CD7)

DEFINITION A rare malignancy (under 1% of all breast cancers) presenting typically in older men as a painless subareolar mass; overwhelmingly hormone receptor-positive, and associated with BRCA2 mutations, Klinefelter syndrome, and conditions causing hyperoestrogenism.

Stage / Setting	Principal Treatment Approach
Localised	Modified radical mastectomy (breast conservation less feasible given limited breast tissue) with sentinel node biopsy
Node-positive/high-risk	Adjuvant chemotherapy and radiotherapy per standard female breast cancer protocols, extrapolated given rarity of dedicated trials
Metastatic (nearly always hormone receptor-positive)	Endocrine therapy (tamoxifen preferred over aromatase inhibitors alone, which are less effective in men without concurrent GnRH suppression) ± targeted therapy

COMPLICATIONS

Disease-related:

- Delayed diagnosis relative to women due to low awareness, often presenting at a more advanced stage
- Nipple retraction/discharge and skin/chest wall invasion given limited surrounding breast tissue
- Associated risk of prostate cancer and other BRCA2-related malignancies
- Axillary nodal metastasis at presentation, more common than in female breast cancer due to delayed diagnosis

Treatment-related:

- Psychosocial distress from a 'female' cancer diagnosis in men, often under-addressed
- Sexual dysfunction and gynaecomastia from endocrine therapy
- Venous thromboembolism on tamoxifen
- Osteoporosis from long-term androgen/oestrogen suppression

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Tamoxifen	First-line endocrine therapy (adjuvant/ metastatic)	20 mg orally once daily	Venous thromboembolism, hot flushes, decreased libido
GnRH Agonist + Aromatase Inhibitor	Alternative endocrine approach if tamoxifen not tolerated	Per standard dosing (e.g. leuprolide monthly + anastrozole 1 mg daily)	Hot flushes, bone loss, loss of libido

★ CLINICAL PEARLS & EXAM FOCUS

▸ Awareness that breast cancer occurs in men prevents diagnostic delay – any man with a new subareolar mass warrants prompt evaluation.

▸ Genetic counselling (BRCA2) should be offered given the strong hereditary association in male breast cancer.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Breast Cancer (current version)

M22 Oesophageal Squamous Cell Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the palliative options for malignant dysphagia in oesophageal carcinoma. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Select appropriate palliative interventional options for luminal GI obstruction (PD3, PD8)

DEFINITION Typically arises in the upper/mid oesophagus, strongly linked to tobacco, alcohol, and in India additionally to hot beverage consumption and nutritional factors; presents with progressive dysphagia (solids before liquids) and weight loss.

Stage / Setting	Principal Treatment Approach
Early/superficial	Endoscopic resection or oesophagectomy for very early disease
Locally advanced, resectable	Neoadjuvant chemoradiation followed by oesophagectomy (trimodality therapy)
Metastatic/unresectable	Palliative chemotherapy ± immunotherapy; oesophageal stenting or feeding jejunostomy for dysphagia palliation

COMPLICATIONS

Disease-related:

- Progressive dysphagia leading to malnutrition and cachexia
- Tracheo-oesophageal fistula from direct tumour invasion, causing aspiration pneumonia
- Massive haemorrhage from aortic or major vessel erosion
- Mediastinal and recurrent laryngeal nerve invasion causing hoarseness

Treatment-related:

- Anastomotic leak after oesophagectomy, a major surgical complication
- Oesophageal stent migration or perforation
- Radiation-induced oesophagitis worsening dysphagia transiently
- Feeding jejunostomy site infection/leakage

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin + 5-Fluorouracil	Neoadjuvant/palliative chemotherapy	Cisplatin 75-100 mg/m² + 5FU 750-1000 mg/m²/day infusion, every 3-4 weeks	Nephrotoxicity, mucositis, myelosuppression
Nivolumab	Adjuvant after trimodality therapy with residual disease, or metastatic second-line	240 mg IV every 2 weeks	Immune-related adverse events

★ CLINICAL PEARLS & EXAM FOCUS

▸ A self-expanding metal stent provides rapid relief of malignant dysphagia and is a key palliative intervention when surgery/chemoradiation is not feasible.

▸ Tracheo-oesophageal fistula is a feared complication – new cough with swallowing ('bovine cough') should prompt urgent evaluation.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Esophageal and Esophagogastric Junction Cancers (current version)

M23 Oesophageal Adenocarcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is the role of HER2 testing in gastro-oesophageal adenocarcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply biomarker-directed therapy in upper GI malignancy (CD4.1, CD2.1)

DEFINITION Typically arises in the lower oesophagus/gastro-oesophageal junction from Barrett's metaplasia secondary to chronic gastro-oesophageal reflux disease; incidence is rising in Western populations and in those with obesity.

Stage / Setting	Principal Treatment Approach
Early (T1a, limited to mucosa)	Endoscopic mucosal resection/submucosal dissection
Locally advanced, resectable	Perioperative chemotherapy (FLOT regimen) or neoadjuvant chemoradiation followed by surgery
Metastatic	HER2 testing to guide trastuzumab addition if positive; otherwise fluoropyrimidine-platinum chemotherapy ± immunotherapy

COMPLICATIONS

Disease-related:

- Progressive dysphagia with aspiration risk
- Upper GI bleeding from a friable gastro-oesophageal junction tumour
- Gastric outlet obstruction if tumour extends into the cardia
- Hepatic and peritoneal metastasis in advanced disease

Treatment-related:

- Anastomotic leak and chylothorax after oesophagectomy
- Dumping syndrome after gastro-oesophageal junction resection
- Reflux oesophagitis in the remaining oesophagus post-surgery
- Cardiotoxicity with trastuzumab-containing regimens

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
FLOT (5FU, Leucovorin, Oxaliplatin, Docetaxel)	Perioperative chemotherapy, resectable disease	Per standard 2-weekly FLOT protocol	Myelosuppression, neuropathy (oxaliplatin), diarrhoea
Trastuzumab	HER2-positive metastatic disease, added to chemotherapy	8 mg/kg loading then 6 mg/kg every 3 weeks	Cardiotoxicity – baseline and serial echocardiography required

★ CLINICAL PEARLS & EXAM FOCUS

► Barrett's oesophagus surveillance is key secondary prevention – a frequently tested public-health point.

► HER2 testing should be routine in metastatic gastro-oesophageal adenocarcinoma, analogous to breast cancer.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Esophageal and Esophagogastric Junction Cancers (current version)

M24 Gastric Adenocarcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is vitamin B12 supplementation required after total gastrectomy? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Anticipate nutritional complications of upper GI cancer surgery (CD5, PD4)

DEFINITION Arises from gastric mucosa, with intestinal (Helicobacter pylori and intestinal metaplasia-associated) and diffuse (including signet-ring cell, associated with CDH1 mutations) histological subtypes; often presents late with vague dyspepsia, weight loss or a palpable epigastric mass.

Stage / Setting	Principal Treatment Approach
Early (mucosal/ submucosal)	Endoscopic resection or gastrectomy with lymphadenectomy
Locally advanced, resectable	Perioperative chemotherapy (FLOT) with gastrectomy and D2 lymphadenectomy
Metastatic	HER2/PD-L1/MSI testing to guide therapy; fluoropyrimidine-platinum backbone ± trastuzumab or immunotherapy

COMPLICATIONS

Disease-related:

- Gastric outlet obstruction causing vomiting and inability to tolerate oral intake
- Peritoneal carcinomatosis with malignant ascites (common mode of spread, especially diffuse/signet-ring histology)
- Upper GI bleeding from an ulcerating tumour
- Krukenberg tumours (ovarian metastasis) in diffuse-type gastric cancer

Treatment-related:

- Anastomotic leak and dumping syndrome after gastrectomy
- Vitamin B12 deficiency after total gastrectomy (loss of intrinsic factor) – requires lifelong supplementation
- Malnutrition requiring enteral/parenteral support
- Peritoneal recurrence despite curative-intent surgery

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
FLOT (5FU, Leucovorin, Oxaliplatin, Docetaxel)	Perioperative chemotherapy	Standard 2-weekly protocol, 4 cycles pre-and post-operatively	Myelosuppression, peripheral neuropathy
Trastuzumab	HER2-positive metastatic disease	8 mg/kg loading then 6 mg/kg every 3 weeks with chemotherapy	Cardiotoxicity

★ CLINICAL PEARLS & EXAM FOCUS

► Lifelong vitamin B12 supplementation is mandatory after total gastrectomy – frequently missed in follow-up.

► Diffuse/signet-ring histology has a distinct, more peritoneal pattern of spread compared with intestinal-type gastric cancer.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Gastric Cancer (current version)

M25 Gastrointestinal Stromal Tumour (GIST)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must intraoperative tumour rupture be avoided in GIST surgery? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Understand targeted therapy principles and treatment interruption risks (CD4.1, CD2.1)

DEFINITION The commonest mesenchymal tumour of the GI tract, arising from the interstitial cells of Cajal, driven in most cases by activating KIT or PDGFRA mutations; behaviour ranges from indolent to frankly malignant based on size, mitotic rate and site.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Complete surgical resection (avoiding tumour rupture); adjuvant imatinib for high-risk features
Locally advanced/borderline resectable	Neoadjuvant imatinib to downsize tumour, followed by surgery
Metastatic/unresectable	Imatinib first-line; sunitinib second-line; regorafenib third-line, guided by mutation status

COMPLICATIONS

Disease-related:

- Acute GI bleeding from a friable, often ulcerated mucosal surface
- Tumour rupture causing peritoneal seeding, a major prognostic adverse event
- Bowel obstruction from a large intraluminal/exophytic mass
- Liver metastases, the commonest site of distant spread

Treatment-related:

- Tumour flare/rebound growth if imatinib is interrupted abruptly
- Fluid retention and periorbital oedema with long-term imatinib
- Hand-foot skin reaction limiting quality of life on sunitinib/regorafenib
- Cardiotoxicity (rare) with long-term tyrosine kinase inhibitor use

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Imatinib	Adjuvant (high-risk) and first-line metastatic disease	400 mg orally once daily (800 mg if exon 9 KIT mutation)	Oedema, GI upset, cytopenias, periorbital swelling
Sunitinib	Imatinib-resistant/intolerant disease	37.5 mg orally once daily (continuous dosing) or 50 mg on 4/2 schedule	Hypertension, hand-foot syndrome, hypothyroidism
Regorafenib	Third-line therapy	160 mg orally once daily, 3 weeks on/1 week off	Hypertension, hand-foot skin reaction, hepatotoxicity

★ CLINICAL PEARLS & EXAM FOCUS

▸ Avoid biopsy-related or intraoperative tumour rupture – it dramatically worsens prognosis by seeding the peritoneum.

▸ Imatinib should not be stopped abruptly even temporarily, given rapid rebound tumour growth reported on interruption.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Gastrointestinal Stromal Tumors (current version)

M26 Small Bowel Adenocarcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What hereditary syndrome should be considered in small bowel adenocarcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Link hereditary cancer syndromes to GI malignancy screening (CD9, CD4.1)

DEFINITION A rare GI malignancy, most often arising in the duodenum, associated with Crohn's disease, coeliac disease and Lynch syndrome; presents late with vague abdominal pain, obstruction or occult GI bleeding due to the small bowel's relatively silent anatomical location.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Segmental small bowel resection or pancreaticoduodenectomy (for duodenal primaries) with lymphadenectomy
Locally advanced	Surgical resection followed by adjuvant fluoropyrimidine-oxaliplatin-based chemotherapy
Metastatic	Fluoropyrimidine-platinum chemotherapy (extrapolated from colorectal cancer regimens, given rarity of dedicated trials)

COMPLICATIONS

Disease-related:

- Small bowel obstruction, often the presenting complication given insidious growth
- Chronic occult GI bleeding and iron-deficiency anaemia
- Perforation, particularly with Crohn's-associated tumours
- Delayed diagnosis leading to presentation with advanced/metastatic disease

Treatment-related:

- Short bowel syndrome/malabsorption after extensive resection
- Anastomotic leak after pancreaticoduodenectomy (for duodenal primaries)
- Oxaliplatin-induced peripheral neuropathy limiting chemotherapy duration
- Nutritional deficiencies requiring long-term dietary support

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
FOLFOX (5FU, Leucovorin, Oxaliplatin)	Adjuvant and metastatic therapy	Standard 2-weekly FOLFOX protocol	Peripheral neuropathy (oxaliplatin), myelosuppression, diarrhoea
Capecitabine	Alternative fluoropyrimidine backbone	1000-1250 mg/m² twice daily, days 1-14 of 21-day cycle	Hand-foot syndrome, diarrhoea

★ CLINICAL PEARLS & EXAM FOCUS

▸ Lynch syndrome screening should be considered in small bowel adenocarcinoma, particularly in younger patients.

▸ Diagnosis is often delayed due to non-specific symptoms – maintain a high index of suspicion in known Crohn's/coeliac disease with new symptoms.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Small Bowel Adenocarcinoma (current version)

M27 Colon Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role of molecular testing in metastatic colon cancer management. [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply biomarker-directed systemic therapy and manage bowel obstruction in colorectal cancer (CD4.1, PD3)

DEFINITION A leading cause of cancer mortality worldwide, arising via the adenoma-carcinoma sequence or, less commonly, the serrated pathway; associated with Lynch syndrome and familial adenomatous polyposis in a minority of cases, with screening colonoscopy an important preventive strategy.

Stage / Setting	Principal Treatment Approach
Stage I-II (localised)	Surgical resection (colectomy) with adjuvant chemotherapy considered for high-risk Stage II or all Stage III disease
Stage III (nodal)	Surgery followed by adjuvant FOLFOX/CAPOX chemotherapy for 3-6 months
Stage IV (metastatic)	Molecular testing (RAS/BRAF/MSI) to guide therapy; chemotherapy ± biologic (anti-EGFR or anti-VEGF) ± resection of oligometastatic liver/lung disease

COMPLICATIONS

Disease-related:

- Large bowel obstruction, sometimes the presenting feature requiring emergency surgery/stenting
- Perforation with faecal peritonitis
- Liver metastases (commonest site via portal venous drainage)
- Malignant ascites and peritoneal carcinomatosis in advanced disease

Treatment-related:

- Anastomotic leak after colectomy
- Bevacizumab-related GI perforation and impaired wound healing (requires a treatment-free interval around surgery)
- Oxaliplatin-induced cumulative peripheral neuropathy
- Stoma-related complications (prolapse, parastomal hernia, skin excoriation) after colostomy

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
FOLFOX (5FU, Leucovorin, Oxaliplatin)	Adjuvant Stage III and metastatic first-line	Standard 2-weekly protocol	Peripheral neuropathy, myelosuppression
Bevacizumab	Added to chemotherapy in metastatic disease	5 mg/kg every 2 weeks (with FOLFOX/FOLFIRI)	Hypertension, bleeding, GI perforation, impaired wound healing
Cetuximab			
RAS/BRAF wild-type	400 mg/m²		Acneiform rash,
metastatic disease, left-	loading then 250		hypomagnesaemia, infusion
sided primary	mg/m² weekly		reactions

★ CLINICAL PEARLS & EXAM FOCUS

► A colonic stent can relieve obstruction and avoid emergency surgery as a bridge to elective resection or for palliation in unresectable disease.

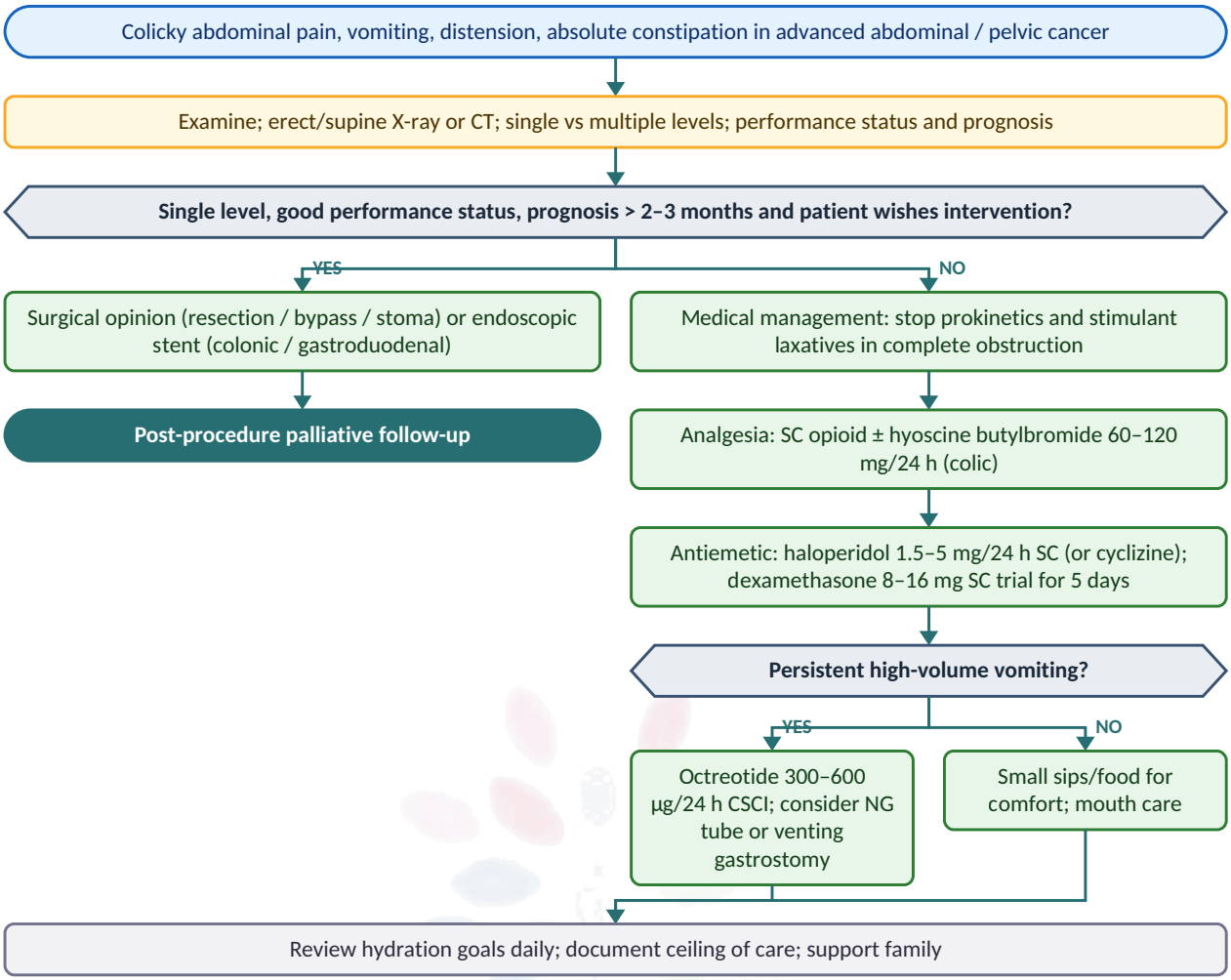
► RAS/BRAF and MSI testing should be performed upfront in metastatic colon cancer – it fundamentally changes the treatment algorithm.

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GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Colon Cancer (current version)

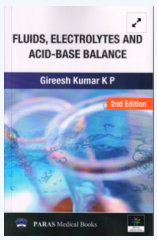
ALGORITHM · Approach to Malignant Bowel Obstruction Algo 4 — full page ›



★ KEY POINTS

- ▸ Metoclopramide is used only in partial (functional) obstruction without colic.
- ▸ Venting PEG frees the patient from a nasogastric tube in refractory obstruction.

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Fluids, Electrolytes & Acid-Base Balance

Clinical reference · 2nd Edition

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M28 Rectal Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss low anterior resection syndrome and its management. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage functional bowel complications after rectal cancer surgery (PD3, PD4)

DEFINITION Adenocarcinoma of the rectum, distinguished from colon cancer by its extraperitoneal location and the resultant emphasis on neoadjuvant therapy and sphincter-preservation considerations in surgical planning.

Stage / Setting	Principal Treatment Approach
Early (T1-T2, N0)	Total mesorectal excision (TME) alone, or local excision for very early/favourable tumours
Locally advanced (T3-T4 or N+)	Neoadjuvant (chemo)radiotherapy or total neoadjuvant therapy followed by TME; non-operative 'watch and wait' in selected complete responders
Metastatic	Systemic chemotherapy ± biologic agents as per colon cancer; consideration of resection for oligometastatic disease

COMPLICATIONS

Disease-related:

- Rectal bleeding and tenesmus from the primary tumour
- Local recurrence in the pelvis, often difficult to salvage surgically
- Fistula formation (rectovaginal/rectovesical) from local invasion
- Obstruction requiring diverting colostomy

Treatment-related:

- Permanent colostomy after abdominoperineal resection for very low tumours
- Low anterior resection syndrome (urgency, frequency, incontinence) after sphincter-preserving surgery
- Sexual and urinary dysfunction from pelvic autonomic nerve injury during TME
- Radiation proctitis and chronic pelvic pain after pelvic radiotherapy

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Capecitabine	Concurrent chemoradiation, radiosensitiser	825 mg/m² twice daily on radiotherapy days	Hand-foot syndrome, diarrhoea
FOLFOX	Total neoadjuvant therapy / adjuvant / metastatic	Standard 2-weekly protocol	Peripheral neuropathy, myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

► Low anterior resection syndrome is common and under-recognised – proactively counsel and manage bowel function expectations after sphincter-preserving surgery.

► 'Watch and wait' after a complete clinical response to neoadjuvant therapy can avoid permanent colostomy in selected patients – an important recent-advances/quality-of-life point.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Rectal Cancer (current version)

M29 Anal Squamous Cell Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is surgery not the primary treatment for anal squamous cell carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Differentiate non-surgical primary treatment paradigms within GI oncology (CD4.1)

DEFINITION Strongly associated with HPV infection (particularly in the setting of HIV co-infection and receptive anal intercourse), arising in the anal canal; unlike most GI malignancies, the primary treatment is non-surgical.

Stage / Setting	Principal Treatment Approach
Localised	Concurrent chemoradiation (Nigro protocol: 5FU-mitomycin with radiotherapy) is primary treatment, with surgery reserved for salvage
Locally advanced	Same chemoradiation approach with close response assessment; abdominoperineal resection if incomplete response/recurrence
Metastatic	Carboplatin-paclitaxel or cisplatin-5FU chemotherapy; immunotherapy for refractory disease

COMPLICATIONS

Disease-related:

- Anal sphincter dysfunction and faecal incontinence from tumour and treatment
- Inguinal lymph node metastasis
- Fistula formation to adjacent pelvic structures
- HIV co-infection complicating immune status and treatment tolerance

Treatment-related:

- Severe perianal radiation dermatitis, often the dose-limiting acute toxicity
- Anal stenosis and chronic pain after chemoradiation
- Delayed, prolonged myelosuppression from mitomycin
- Chronic diarrhoea/faecal urgency from radiation proctitis

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Mitomycin C + 5-Fluorouracil	Concurrent with radiotherapy (Nigro protocol)	Mitomycin 10 mg/m² day 1 + 5FU 1000 mg/m²/day days 1-4, with radiotherapy	Myelosuppression (mitomycin can cause delayed/prolonged cytopenia), mucositis
Carboplatin + Paclitaxel	Metastatic disease	Carboplatin AUC 5 + paclitaxel 175 mg/m² every 3-4 weeks	Myelosuppression, peripheral neuropathy

★ CLINICAL PEARLS & EXAM FOCUS

► Anal cancer is primarily treated with chemoradiation, not upfront surgery – a frequently confused point given its GI location.

► HPV vaccination has a role in primary prevention – relevant to public-health discussion in exams.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Anal Carcinoma (current version)

M30 Pancreatic Ductal Adenocarcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is pancreatic cancer associated with a high risk of venous thromboembolism? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Integrate early palliative care in a rapidly fatal GI malignancy (CD4.1, AD2, PD3)

DEFINITION One of the most lethal solid malignancies, arising from pancreatic ductal epithelium, typically presenting late with painless jaundice (head of pancreas), back pain, or new-onset diabetes; resectability at diagnosis is the single biggest prognostic determinant.

Stage / Setting	Principal Treatment Approach
Resectable	Surgery (Whipple procedure for head lesions, distal pancreatectomy for body/tail) followed by adjuvant chemotherapy (FOLFIRINOX or gemcitabine-capecitabine)
Borderline resectable/ locally advanced	Neoadjuvant chemotherapy ± chemoradiation, with reassessment for resection
Metastatic	FOLFIRINOX (good performance status) or gemcitabine-nab-paclitaxel (poorer performance status); strong early integration of palliative care is standard

COMPLICATIONS

Disease-related:

- Obstructive jaundice from biliary obstruction, requiring stenting
- Duodenal/gastric outlet obstruction from local invasion
- Malignant ascites and peritoneal carcinomatosis
- Thromboembolism – pancreatic cancer carries one of the highest rates of cancer-associated VTE
- Exocrine pancreatic insufficiency causing malabsorption and steatorrhoea

Treatment-related:

- Biliary stent occlusion/cholangitis requiring re-intervention
- Delayed gastric emptying after Whipple procedure
- Pancreatic fistula, a major post-Whipple surgical complication
- Severe, often refractory pain requiring coeliac plexus block in addition to systemic opioids

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
FOLFIRINOX (5FU, Leucovorin, Irinotecan, Oxaliplatin)	Fit patients, adjuvant and metastatic	Standard 2-weekly protocol (often dose-modified)	Severe myelosuppression, diarrhoea, peripheral neuropathy – requires good performance status
Gemcitabine + Nab-Paclitaxel	Metastatic, less fit patients	Gemcitabine 1000 mg/m² + nab-paclitaxel 125 mg/m², days 1, 8, 15 of 28-day cycle	Myelosuppression, peripheral neuropathy, pneumonitis

★ CLINICAL PEARLS & EXAM FOCUS

► Given the high VTE risk, a low threshold for prophylactic/therapeutic anticoagulation is warranted unless contraindicated.

► Early palliative care referral at diagnosis improves quality of life and is now a guideline-endorsed standard, not an afterthought, in pancreatic cancer.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Pancreatic Adenocarcinoma (current version)

M31 Pancreatic Neuroendocrine Tumour

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the hormonal syndromes associated with functioning pancreatic neuroendocrine tumours. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and manage hormone-secreting tumour syndromes (CD4.1, CD9.1)

DEFINITION Arises from pancreatic islet cells, may be functioning (secreting insulin, gastrin, glucagon, VIP) causing distinct hormonal syndromes, or non-functioning (the majority, presenting with mass effect); generally more indolent than ductal adenocarcinoma.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Surgical resection (enucleation for small functioning tumours, formal resection for larger/non-functioning tumours)
Unresectable/metastatic, low-grade	Somatostatin analogues for symptom and modest antiproliferative control; liver-directed therapy for liver-dominant disease
Progressive/higher-grade metastatic	Everolimus or sunitinib (targeted therapy); peptide receptor radionuclide therapy (PRRT) for somatostatin-receptor-positive disease; chemotherapy for high-grade tumours

COMPLICATIONS

Disease-related:

- Hypoglycaemic episodes (insulinoma) or severe peptic ulceration (gastrinoma/Zollinger-Ellison syndrome)
- Necrolytic migratory erythema and diabetes (glucagonoma)
- Watery diarrhoea, hypokalaemia and achlorhydria (VIPoma/WDHA syndrome)
- Liver metastases, the dominant site and driver of hormonal symptom burden

Treatment-related:

- Hormonal crisis precipitated by biopsy/surgical manipulation of a functioning tumour without adequate pre-treatment cover
- Pneumonitis and stomatitis with everolimus
- Cholelithiasis with long-term somatostatin analogue use
- Treatment-related hypothyroidism with sunitinib

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Octreotide LAR	Functioning tumour symptom control, antiproliferative in low-grade disease	20-30 mg IM every 4 weeks	Gallstones, hyperglycaemia/hypoglycaemia fluctuation
Everolimus	Progressive, well-differentiated metastatic disease	10 mg orally once daily	Pneumonitis, stomatitis, hyperglycaemia, immunosuppression-related infection risk
Sunitinib	Progressive metastatic disease, alternative to everolimus	37.5 mg orally once daily	Hypertension, hand-foot syndrome, hypothyroidism

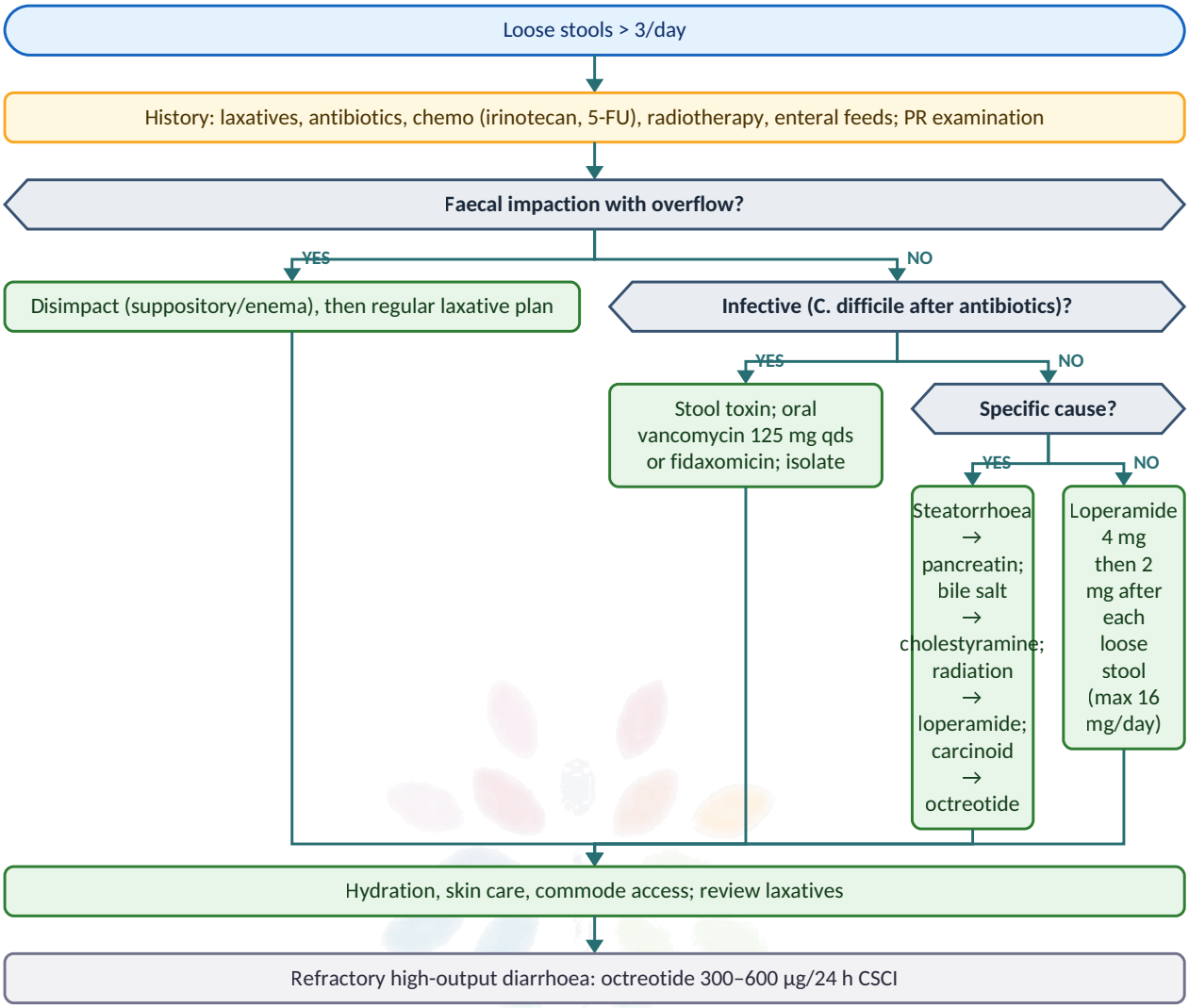
CLINICAL PEARLS & EXAM FOCUS

- Identify and biochemically characterise functioning syndromes (insulin, gastrin, glucagon, VIP) before intervention to anticipate and prevent hormonal crises.
- PRRT is an important, guideline-endorsed option for somatostatin-receptor-positive progressive disease – a recent-advances exam favourite.

GUIDELINES & RECENT ADVANCES

•	NANETS/ENETS Consensus Guidelines: Pancreatic Neuroendocrine Tumours		

ALGORITHM · Diarrhoea in Palliative Care Algo 30 — full page ›



★ KEY POINTS

- ▸ Never start loperamide without excluding impaction and C. difficile.
- ▸ Irinotecan early diarrhoea is cholinergic — atropine responds.

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M32 Hepatocellular Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must liver function, not just tumour stage, guide HCC treatment selection? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Integrate chronic liver disease management with oncological decision-making (CD4.1, CD5)

DEFINITION The commonest primary liver malignancy, arising almost always in the context of chronic liver disease (viral hepatitis B/C, alcohol-related cirrhosis, non-alcoholic fatty liver disease), making underlying liver function as important as tumour burden in determining treatment options.

Stage / Setting	Principal Treatment Approach
Early stage, preserved liver function	Surgical resection or liver transplantation (if within Milan criteria); radiofrequency/microwave ablation for small tumours
Intermediate stage	Transarterial chemoembolization (TACE) for multinodular, liver-confined disease
Advanced stage/ extrahepatic spread	Systemic therapy – atezolizumab-bevacizumab first-line, or tyrosine kinase inhibitors (sorafenib/ lenvatinib) if immunotherapy contraindicated

COMPLICATIONS

Disease-related:

- Decompensation of underlying cirrhosis (ascites, encephalopathy, variceal bleeding) often dominating the clinical picture more than tumour bulk
- Tumour rupture causing haemoperitoneum, a surgical emergency
- Portal vein thrombosis/tumour thrombus worsening portal hypertension
- Paraneoplastic hypoglycaemia or erythrocytosis

Treatment-related:

- Post-embolisation syndrome (fever, pain, nausea) after TACE
- Variceal bleeding precipitated or unmasked by bevacizumab's antiangiogenic effect
- Hepatic decompensation triggered by systemic therapy in marginal liver function
- Hand-foot skin reaction limiting quality of life on tyrosine kinase inhibitors

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Atezolizumab + Bevacizumab	First-line advanced HCC, preserved liver function (Child-Pugh A)	Atezolizumab 1200 mg + bevacizumab 15 mg/ kg IV every 3 weeks	Immune-related toxicity, bleeding (bevacizumab) – requires endoscopic variceal screening first
Sorafenib	Alternative first-line or if immunotherapy contraindicated	400 mg orally twice daily	Hand-foot skin reaction, hypertension, diarrhoea

★ CLINICAL PEARLS & EXAM FOCUS

► Treatment choice in HCC must weigh BOTH tumour stage AND underlying liver function (Child-Pugh class) – a tumour-only staging approach is a common exam error.

► Variceal screening and management should precede bevacizumab-containing regimens given bleeding risk.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Hepatobiliary Cancers (current version)

M33 Intrahepatic Cholangiocarcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What molecular alterations are targetable in intrahepatic cholangiocarcinoma? [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply molecular profiling to guide second-line therapy in biliary tract cancer (CD4.1, CD2.1)

DEFINITION Adenocarcinoma arising from the intrahepatic bile ducts, increasingly recognised with rising incidence, associated with liver fluke infestation (endemic areas), primary sclerosing cholangitis, and hepatolithiasis; often presents with a liver mass rather than jaundice (unlike extrahepatic/hilar tumours).

Stage / Setting	Principal Treatment Approach
Localised, resectable	Hepatic resection with lymphadenectomy; adjuvant capecitabine for resected disease
Locally advanced/ unresectable	Locoregional therapy (TACE, radioembolization) or systemic chemotherapy
Metastatic	Gemcitabine-cisplatin chemotherapy ± durvalumab first-line; targeted therapy (FGFR2 fusion, IDH1 mutation inhibitors) if molecularly eligible on progression

COMPLICATIONS

Disease-related:

- Biliary obstruction with cholangitis, often recurrent
- Intrahepatic spread with multifocal liver involvement
- Peritoneal carcinomatosis
- Hepatic failure from extensive parenchymal replacement

Treatment-related:

- Recurrent cholangitis requiring repeated biliary drainage procedures
- Hepatic insufficiency after extensive liver resection
- Hyperphosphataemia and ocular toxicity with FGFR inhibitors
- Biliary-enteric anastomotic stricture after surgery

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Gemcitabine + Cisplatin + Durvalumab	First-line metastatic/ advanced disease	Gemcitabine 1000 mg/m² + cisplatin 25 mg/m² days 1,8 + durvalumab 1500 mg, every 3 weeks	Myelosuppression, nephrotoxicity, immune-related toxicity
Pemigatinib	FGFR2 fusion-positive disease, second-line	13.5 mg orally once daily (2 weeks on/1 week off)	Hyperphosphataemia, retinal detachment risk, dry mouth

★ CLINICAL PEARLS & EXAM FOCUS

▸ Molecular profiling (FGFR2, IDH1) should be performed in intrahepatic cholangiocarcinoma, as it opens targeted therapy options unavailable in extrahepatic disease.

▸ Recurrent, unexplained cholangitis in an at-risk patient (liver fluke exposure, PSC) should prompt imaging to exclude cholangiocarcinoma.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Hepatobiliary Cancers (current version)

M34 Gallbladder Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What complication can follow laparoscopic cholecystectomy for unsuspected gallbladder cancer? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise incidental malignancy and plan appropriate re-intervention (CD4.1, PD4)

DEFINITION An aggressive biliary tract malignancy with marked geographic variation (notably higher incidence in parts of North India and South America), strongly associated with gallstones and chronic cholecystitis; often diagnosed incidentally on cholecystectomy specimens or presents late with jaundice/mass.

Stage / Setting	Principal Treatment Approach
Incidental, early (T1a) on cholecystectomy specimen	Simple cholecystectomy alone may be curative
Incidental T1b or higher, or clinically detected resectable disease	Radical cholecystectomy (liver resection + lymphadenectomy) ± bile duct resection
Locally advanced/metastatic	Gemcitabine-cisplatin chemotherapy ± durvalumab; biliary drainage for obstructive jaundice

COMPLICATIONS

Disease-related:

- Obstructive jaundice from direct invasion of the common bile duct
- Direct liver invasion (gallbladder's anatomical proximity)
- Duodenal/gastric outlet obstruction from local extension
- Peritoneal and port-site metastasis after laparoscopic cholecystectomy for unsuspected cancer

Treatment-related:

- Port-site recurrence after laparoscopic cholecystectomy for an unsuspected malignancy, a well-recognised complication prompting a move to open radical surgery when suspected pre-operatively
- Biliary stent occlusion and cholangitis
- Hepatic insufficiency after extended liver resection
- Delayed diagnosis given non-specific presenting symptoms

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Gemcitabine + Cisplatin + Durvalumab	First-line advanced/ metastatic disease	Gemcitabine 1000 mg/m² + cisplatin 25 mg/m² days 1,8 + durvalumab 1500 mg, every 3 weeks	Myelosuppression, nephrotoxicity, immune-related toxicity

★ CLINICAL PEARLS & EXAM FOCUS

► Incidental gallbladder cancer found on routine cholecystectomy should prompt staging and consideration of radical re-resection depending on T-stage.

► Any suspicious gallbladder wall thickening or mass on imaging warrants pre-operative planning for open (not laparoscopic) surgery to avoid port-site seeding.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Hepatobiliary Cancers (current version)

M35 Appendiceal Tumours (Neuroendocrine & Pseudomyxoma Peritonei)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the management options for pseudomyxoma peritonei. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Manage progressive, non-curable peritoneal malignancy with a palliative, staged approach (CD4.1, CD3.1)

DEFINITION A heterogeneous group including appendiceal neuroendocrine (carcinoid) tumours, usually indolent and found incidentally after appendicectomy, and mucinous neoplasms that can rupture to cause pseudomyxoma peritonei – diffuse mucinous peritoneal deposits causing progressive abdominal distension.

Stage / Setting	Principal Treatment Approach
Small appendiceal carcinoid (<1-2 cm), incidental	Simple appendicectomy is usually curative
Larger carcinoid or mucinous neoplasm with peritoneal spread	Right hemicolectomy ± cytoreductive surgery with hyperthermic intraperitoneal chemotherapy (HIPEC) for pseudomyxoma peritonei
Diffuse/recurrent pseudomyxoma peritonei	Repeat cytoreductive surgery with HIPEC where feasible; otherwise symptom-directed palliative management of progressive distension

COMPLICATIONS

Disease-related:

- Progressive abdominal distension from accumulating mucinous ascites (pseudomyxoma peritonei)
- Bowel obstruction from diffuse peritoneal mucinous deposits
- Appendiceal perforation precipitating peritoneal mucin dissemination
- Nutritional compromise from recurrent distension and early satiety

Treatment-related:

- Major morbidity (anastomotic leak, prolonged ileus, sepsis) after extensive cytoreductive surgery with HIPEC
- Recurrent ascites/mucin accumulation despite surgery, requiring repeated drainage
- Short bowel syndrome after extensive bowel resection during cytoreduction
- Adhesive bowel obstruction from multiple prior laparotomies

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Mitomycin C	Intraperitoneal chemotherapy component of HIPEC	Per HIPEC protocol (intraoperative, heated intraperitoneal administration)	Myelosuppression, anastomotic healing impairment
Octreotide LAR	Symptomatic appendiceal carcinoid with carcinoid syndrome (rare)	20-30 mg IM every 4 weeks	Gallstones, GI discomfort

★ CLINICAL PEARLS & EXAM FOCUS

▸ Pseudomyxoma peritonei progresses slowly but relentlessly – repeated, carefully timed cytoreductive surgery can offer meaningful palliation and prolonged survival in selected patients.

▸ An appendiceal mass found incidentally at appendicectomy should always be sent for histopathology – carcinoid vs mucinous neoplasm changes subsequent management entirely.

GUIDELINES & RECENT ADVANCES

- Peritoneal Surface Oncology Group International (PSOGI) Consensus on Pseudomyxoma Peritonei

M36 Renal Cell Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the paraneoplastic syndromes associated with renal cell carcinoma. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise paraneoplastic manifestations of genitourinary malignancy (CD4.1, CD9.1)

DEFINITION Arises from renal tubular epithelium, classically presenting with the triad of haematuria, flank pain and a palpable mass (now uncommon as most are detected incidentally on imaging); clear-cell histology is commonest and associated with VHL pathway alterations.

Stage / Setting	Principal Treatment Approach
Localised	Partial or radical nephrectomy depending on tumour size/location; active surveillance for small incidental masses in frail patients
Locally advanced	Radical nephrectomy ± adjuvant immunotherapy (pembrolizumab) for high-risk resected disease
Metastatic	Risk-stratified combination therapy – immunotherapy doublet (nivolumab-ipilimumab) or immunotherapy plus tyrosine kinase inhibitor (pembrolizumab-axitinib)

COMPLICATIONS

Disease-related:

- Tumour thrombus extending into the renal vein and inferior vena cava, occasionally reaching the right atrium
- Paraneoplastic syndromes – hypercalcaemia, erythrocytosis, Stauffer syndrome (non-metastatic hepatic dysfunction)
- Bone and lung metastases, the commonest distant sites
- Solitary late metastases (even 10+ years after nephrectomy), a distinctive feature of RCC

Treatment-related:

- Immune-related colitis/endocrinopathy/pneumonitis with checkpoint inhibitor combinations
- Hypertension and proteinuria with VEGF-pathway tyrosine kinase inhibitors
- Loss of renal function after nephrectomy, particularly in patients with pre-existing CKD
- IVC thrombus-related complications (pulmonary embolism) if tumour thrombus dislodges

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Nivolumab + Ipilimumab	First-line, intermediate/ poor-risk metastatic disease	Nivolumab 3 mg/kg + ipilimumab 1 mg/ kg every 3 weeks x4, then nivolumab maintenance	Immune-related adverse events, can be severe
Pembrolizumab + Axitinib	First-line, across risk groups	Pembrolizumab 200 mg every 3 weeks + axitinib 5 mg twice daily	Hypertension, diarrhoea, immune-related toxicity, hand-foot syndrome

★ CLINICAL PEARLS & EXAM FOCUS

► RCC can present with late, solitary metastases many years after apparently curative nephrectomy – lifelong surveillance has some rationale, unlike most solid tumours.

► Paraneoplastic erythrocytosis/hypercalcaemia should prompt renal imaging even without classic urinary symptoms.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Kidney Cancer (current version)

M37 Urothelial (Bladder) Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What clinical presentation should always raise suspicion of bladder carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Evaluate painless haematuria and select appropriate bladder-sparing vs radical therapy (CD4.1, PD3)

DEFINITION The commonest bladder malignancy, strongly linked to tobacco smoking and occupational aromatic amine exposure, typically presenting with painless haematuria; behaviour ranges from superficial/non-muscle-invasive (recurrent but rarely lethal) to muscle-invasive (requiring aggressive therapy).

Stage / Setting	Principal Treatment Approach
Non-muscle-invasive	Transurethral resection of bladder tumour (TURBT) followed by intravesical BCG immunotherapy for high-risk disease
Muscle-invasive, localised	Neoadjuvant cisplatin-based chemotherapy followed by radical cystectomy with urinary diversion, or bladder-preservation trimodality therapy
Metastatic	Platinum-based chemotherapy first-line, followed by maintenance avelumab (immunotherapy) if disease control achieved; enfortumab vedotin-pembrolizumab increasingly used first-line

COMPLICATIONS

Disease-related:

- Recurrent gross haematuria causing clot retention and anaemia
- Hydronephrosis from ureteric orifice obstruction
- Local invasion into pelvic organs (rectum, prostate, vagina) in muscle-invasive disease
- Distant metastasis to lung, liver, bone in advanced disease

Treatment-related:

- Stoma/urinary diversion complications (ileal conduit leak, stenosis, pouchitis) after cystectomy
- Systemic BCG infection requiring antitubercular therapy, a rare but serious complication
- Radiation cystitis in bladder-preservation approaches
- Peripheral neuropathy from enfortumab vedotin, sometimes dose-limiting

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Intravesical BCG	Adjuvant therapy, high-risk non-muscle-invasive disease	Standard induction (6 weekly instillations) then maintenance schedule	Cystitis symptoms, rarely systemic BCG infection ('BCGosis') requiring antitubercular therapy
Enfortumab Vedotin + Pembrolizumab	First-line metastatic disease	Per approved combination schedule	Peripheral neuropathy, skin reactions, hyperglycaemia, immune-related toxicity

★ CLINICAL PEARLS & EXAM FOCUS

▸ Painless gross haematuria in a smoker over 40 is bladder cancer until proven otherwise – a classic exam red flag.

▸ Bladder preservation (trimodality therapy) is an appropriate alternative to cystectomy in well-selected patients wishing to avoid a stoma.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Bladder Cancer (current version)

M38 Upper Tract Urothelial Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is adjuvant chemotherapy often limited after nephroureterectomy? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Anticipate renal function decline affecting systemic therapy eligibility (CD4.1, CD5)

DEFINITION Urothelial carcinoma arising in the renal pelvis or ureter, sharing risk factors with bladder cancer but behaving somewhat more aggressively at presentation due to the thinner wall of the upper urinary tract; associated with Lynch syndrome in a notable minority.

Stage / Setting	Principal Treatment Approach
Localised, low-grade	Kidney-sparing endoscopic resection/ablation in selected low-risk, low-grade tumours
Localised, high-grade/ invasive	Radical nephroureterectomy with bladder cuff excision
Metastatic	Platinum-based chemotherapy, followed by maintenance immunotherapy, analogous to bladder cancer

COMPLICATIONS

Disease-related:

- Hydronephrosis and renal function decline from ureteric obstruction
- Intraluminal seeding ('tumour spill') down the ureter and into the bladder
- Bilateral disease or metachronous bladder tumours given field-cancerisation of urothelium
- Flank pain and haematuria from the primary lesion

Treatment-related:

- Post-nephroureterectomy chronic kidney disease, limiting subsequent cisplatin-based chemotherapy eligibility
- Anastomotic/ureteric stricture after kidney-sparing endoscopic management
- Bladder recurrence after nephroureterectomy requiring ongoing cystoscopic surveillance
- Reduced renal reserve complicating safe chemotherapy dosing

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Gemcitabine + Cisplatin	First-line metastatic/ adjuvant high-risk disease	Standard 21-28 day protocol	Myelosuppression, nephrotoxicity (caution given frequent baseline renal impairment post-nephroureterectomy)

★ CLINICAL PEARLS & EXAM FOCUS

► Lynch syndrome screening should be considered, particularly in younger patients or those with a suggestive family history.

► Renal function after nephroureterectomy often falls below the threshold for cisplatin – carboplatin-based regimens are frequently necessary in practice despite being less effective.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Bladder Cancer (Upper Tract Urothelial Carcinoma section, current version)



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M39 Prostate Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must an antiandrogen be co-administered when starting a GnRH agonist for prostate cancer? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Anticipate and prevent the testosterone flare phenomenon in androgen deprivation therapy (CD4.1, PD2)

DEFINITION The commonest male genitourinary malignancy, typically slow-growing and often diagnosed via PSA screening or incidentally; treatment intensity is carefully matched to risk group given the generally indolent course of localised, low-risk disease.

Stage / Setting	Principal Treatment Approach
Low-risk, localised	Active surveillance is often appropriate given very low risk of progression over many years
Intermediate/high-risk, localised	Radical prostatectomy or definitive radiotherapy with androgen deprivation therapy
Metastatic (hormone-sensitive)	Androgen deprivation therapy combined with an androgen-receptor pathway inhibitor (abiraterone/enzalutamide) or docetaxel

COMPLICATIONS

Disease-related:

- Bone metastases causing pain, pathological fracture and spinal cord compression (axial skeleton predilection)
- Bladder outlet obstruction and urinary retention from local prostatic growth
- Hydronephrosis from ureteric obstruction by local extension or nodal disease
- Progression from hormone-sensitive to castration-resistant disease, changing management entirely

Treatment-related:

- Osteoporosis and fracture risk from long-term androgen deprivation therapy
- Hot flushes, sexual dysfunction and metabolic syndrome (weight gain, insulin resistance) from ADT
- Testosterone flare phenomenon at GnRH agonist initiation, transiently worsening bone pain/obstruction unless covered by an antiandrogen
- Hepatotoxicity and fluid retention with abiraterone

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Leuprolide (GnRH agonist)	Androgen deprivation therapy, all metastatic stages	3.75-45 mg IM depot every 1-6 months depending on formulation	Hot flushes, osteoporosis, cardiovascular risk, initial testosterone flare (co-administer antiandrogen)
Abiraterone + Prednisone	Metastatic hormone-sensitive or castration-resistant disease	1000 mg abiraterone once daily + prednisone 5 mg once/ twice daily	Hepatotoxicity, hypertension, hypokalaemia – monitor liver function and electrolytes

★ CLINICAL PEARLS & EXAM FOCUS

► Always co-administer an antiandrogen (e.g. bicalutamide) for the first weeks of GnRH agonist therapy to prevent the testosterone flare from worsening bone pain or causing cord compression.

► Back pain in a man with known prostate cancer should prompt urgent evaluation for spinal metastasis/ cord compression given the strong osteotropism of this cancer.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Prostate Cancer (current version)

M40 Testicular Seminoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why should sperm banking be discussed before treating testicular cancer? [Viva, ethics]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply fertility-preservation counselling in young oncology patients (AD5, PD2)

DEFINITION A germ cell tumour of young men (peak incidence 25-45 years), typically presenting as a painless testicular mass; highly chemo- and radiosensitive, with excellent cure rates even in advanced disease.

Stage / Setting	Principal Treatment Approach
Stage I	Radical inguinal orchidectomy followed by surveillance (preferred) or adjuvant carboplatin/ radiotherapy
Stage II (nodal)	Orchidectomy followed by radiotherapy (bulky-independent small volume) or chemotherapy (bulkier disease)
Stage III (metastatic)	Orchidectomy followed by cisplatin-based combination chemotherapy (BEP regimen)

COMPLICATIONS

Disease-related:

- Retroperitoneal lymph node metastasis causing back pain or ureteric obstruction
- Rarely, pulmonary/mediastinal metastasis in advanced disease
- Contralateral testicular germ cell neoplasia in situ (risk of second primary)
- Gynaecomastia from beta-hCG-related oestrogenic effect in some cases

Treatment-related:

- Bleomycin-induced pulmonary fibrosis, a serious and sometimes irreversible toxicity requiring baseline and interval pulmonary function monitoring
- Infertility from both the orchidectomy and chemotherapy – sperm banking should be offered before treatment
- Long-term cardiovascular and metabolic risk from cisplatin-based chemotherapy in young survivors
- Secondary malignancy risk (leukaemia) from chemotherapy, a rare late effect

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Bleomycin + Etoposide + Cisplatin (BEP)	Metastatic/advanced disease chemotherapy	Standard 21-day BEP protocol for 3-4 cycles	Bleomycin pulmonary toxicity, cisplatin nephro/ototoxicity, myelosuppression
Carboplatin	Adjuvant therapy, Stage I disease	AUC 7, single dose or 1-2 cycles	Myelosuppression, generally well tolerated compared with BEP

★ CLINICAL PEARLS & EXAM FOCUS

▸ Sperm banking must be offered before any treatment given young patient age and treatment-related infertility risk – a frequently tested communication/consent point.

▸ Testicular seminoma has one of the best cure rates in solid oncology even with metastatic disease – important for accurate patient counselling.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Testicular Cancer (current version)

M41 Non-Seminomatous Testicular Germ Cell Tumour

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is growing teratoma syndrome and why is surgery, not chemotherapy, indicated? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Distinguish chemoresponsive from chemoresistant elements within a germ cell tumour (CD4.1, CD2.1)

DEFINITION A heterogeneous group (embryonal carcinoma, yolk sac tumour, choriocarcinoma, teratoma) often presenting with more aggressive behaviour and higher tumour marker elevation (AFP, beta-hCG) than seminoma; retroperitoneal lymph node dissection plays a larger surgical role.

Stage / Setting	Principal Treatment Approach
Stage I	Orchidectomy followed by surveillance, primary retroperitoneal lymph node dissection, or adjuvant chemotherapy depending on risk features
Stage II-III	Orchidectomy followed by cisplatin-based combination chemotherapy (BEP), with post-chemotherapy retroperitoneal lymph node dissection for residual masses
Poor-risk/relapsed disease	Intensified or salvage chemotherapy regimens (VeIP, TIP) ± high-dose chemotherapy with autologous stem cell rescue

COMPLICATIONS

Disease-related:

- Rapidly rising tumour markers (AFP, beta-hCG) indicating aggressive/bulky disease
- Haemorrhage into choriocarcinoma metastases (lung, brain) – a distinctive and dangerous complication
- Retroperitoneal nodal masses causing ureteric obstruction or vascular compression
- Growing teratoma syndrome – enlarging masses despite normalising tumour markers and chemoresponse

Treatment-related:

- Post-chemotherapy retroperitoneal lymph node dissection complications (retrograde ejaculation, lymphatic leak, bowel/vascular injury)
- Bleomycin pulmonary toxicity, particularly with pre-existing lung disease or high cumulative dose
- Growing teratoma syndrome requiring surgical resection rather than more chemotherapy (chemo-resistant)
- Long-term nephrotoxicity and ototoxicity from cisplatin-based regimens

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Bleomycin + Etoposide + Cisplatin (BEP)	First-line chemotherapy, all risk groups	Standard 21-day BEP protocol for 3-4 cycles	Pulmonary toxicity (bleomycin), nephro/ ototoxicity (cisplatin), myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

► Growing teratoma syndrome should be recognised – an enlarging mass with normal/falling markers after chemotherapy needs surgery, not more chemotherapy, since mature teratoma is chemoresistant.

► Intracranial haemorrhage from choriocarcinoma brain metastases can be the presenting emergency – consider this diagnosis in a young man with sudden neurological deficit.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Testicular Cancer (current version)

M42 Penile Squamous Cell Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why does diagnostic delay commonly occur in penile carcinoma, and what is its impact? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Address stigma-related delay and psychosexual distress in genital malignancy (AD6, CD7)

DEFINITION A rare malignancy strongly associated with HPV infection, phimosis and poor genital hygiene, with lower incidence in circumcised populations; presents as a non-healing penile lesion, often with significant diagnostic delay due to embarrassment and stigma.

Stage / Setting	Principal Treatment Approach
Localised, low-stage	Organ-preserving surgery (wide local excision, glansectomy) with penile-sparing intent where feasible
Locally advanced/ nodal	Partial or total penectomy with inguinal lymphadenectomy; neoadjuvant chemotherapy for bulky nodal disease
Metastatic	Platinum-based combination chemotherapy; limited evidence base given rarity

COMPLICATIONS

Disease-related:

- Inguinal lymph node metastasis, often the dominant prognostic factor
- Local destruction of penile tissue causing pain, bleeding and malodour
- Urethral obstruction/fistula from local invasion
- Significant psychosexual and body-image distress

Treatment-related:

- Lymphoedema of the lower limbs/genitalia after inguinal lymphadenectomy
- Wound infection/necrosis after inguinal lymph node dissection, historically a major morbidity
- Psychosexual dysfunction and altered body image after partial/total penectomy
- Diagnostic/treatment delay from patient embarrassment, worsening outcomes

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin + 5-Fluorouracil	Metastatic/neoadjuvant for bulky nodal disease	Cisplatin 75-100 mg/m² + 5FU infusion, every 3-4 weeks	Nephrotoxicity, mucositis, myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

► Diagnostic delay due to stigma is a major, modifiable contributor to poor outcomes – sensitive, non-judgemental communication is essential.

► Inguinal nodal status is the single strongest prognostic factor – careful nodal staging and management should not be deprioritised relative to the primary lesion.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Penile Cancer (current version)

M43 Cervical Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the public-health impact of HPV vaccination on cervical cancer. [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply preventive oncology principles to gynaecological malignancy (AD9, CD9.1)

DEFINITION Almost universally caused by persistent high-risk HPV infection (particularly types 16 and 18), making it one of the most preventable cancers through vaccination and screening; remains a leading cause of cancer death among women in resource-limited settings including much of India.

Stage / Setting	Principal Treatment Approach
Early (Stage IA-IB1/IIA1)	Radical hysterectomy with pelvic lymphadenectomy, or fertility-sparing trachelectomy in selected young patients
Locally advanced (Stage IB2-IVA)	Concurrent chemoradiation (cisplatin-based) with brachytherapy boost is standard of care
Metastatic/recurrent	Platinum-based chemotherapy with bevacizumab ± pembrolizumab (PD-L1 positive)

COMPLICATIONS

Disease-related:

- Ureteric obstruction and hydronephrosis from parametrial invasion, a common cause of renal failure in advanced disease
- Vesicovaginal or rectovaginal fistula from local tumour invasion or treatment
- Vaginal bleeding, sometimes massive, requiring vaginal packing or emergency embolisation
- Pelvic/leg lymphoedema from nodal disease or lymphadenectomy

Treatment-related:

- Radiation proctitis, cystitis and vaginal stenosis after pelvic radiotherapy
- Bevacizumab-associated fistula formation, particularly in previously irradiated tissue
- Lymphoedema of the lower limb after pelvic lymphadenectomy
- Premature ovarian failure/infertility after radical surgery or radiotherapy in young women

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Radiosensitiser during concurrent chemoradiation	40 mg/m² weekly during radiotherapy	Nephrotoxicity, nausea, myelosuppression
Bevacizumab	Added to chemotherapy, metastatic/recurrent disease	15 mg/kg IV every 3 weeks	Bleeding, fistula formation (notably vesicovaginal/rectovaginal), hypertension

★ CLINICAL PEARLS & EXAM FOCUS

► HPV vaccination and cervical screening (Pap smear/HPV testing) are the most impactful public-health interventions in oncology – always highlight this preventive angle.

► Bilateral hydronephrosis in cervical cancer is an ominous sign of advanced parametrial disease and a frequent cause of uraemic death if untreated.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Cervical Cancer (current version)

M44 Endometrial Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why does endometrial cancer often present at an early, curable stage? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Link symptom-based early detection to favourable gynaecological cancer outcomes (AD9, CD4.1)

DEFINITION The commonest gynaecological malignancy in high-resource settings, typically presenting early with postmenopausal bleeding, allowing diagnosis at a curable stage in most cases; strongly linked to unopposed oestrogen exposure, obesity, and Lynch syndrome in a subset.

Stage / Setting	Principal Treatment Approach
Early, low-risk (Stage I, endometrioid, low-grade)	Total hysterectomy with bilateral salpingo-oophorectomy, often without adjuvant therapy
Intermediate/high-risk	Surgery followed by adjuvant vaginal brachytherapy ± pelvic radiotherapy ± chemotherapy
Advanced/metastatic or high-grade/ serous histology	Chemotherapy (carboplatin-paclitaxel) ± immunotherapy, guided by mismatch-repair/microsatellite status

COMPLICATIONS

Disease-related:

- Postmenopausal bleeding, usually the presenting (and early-warning) symptom
- Myometrial and cervical stromal invasion predicting nodal spread risk
- Peritoneal/omental spread in high-grade serous or clear-cell histology, behaving more like ovarian cancer
- Lymphoedema from lymphadenectomy

Treatment-related:

- Pelvic/vaginal radiation-related cystitis, proctitis and vaginal stenosis
- Lymphoedema of the lower limb after lymphadenectomy
- Chemotherapy-related peripheral neuropathy
- Surgical menopause symptoms after bilateral salpingo-oophorectomy in younger patients

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Carboplatin + Paclitaxel	Adjuvant high-risk and metastatic disease	Carboplatin AUC 5-6 + paclitaxel 175 mg/m² every 3 weeks	Myelosuppression, peripheral neuropathy
Pembrolizumab + Lenvatinib	Advanced disease, mismatch-repair-proficient, after prior therapy	Pembrolizumab 200 mg every 3 weeks + lenvatinib 20 mg daily	Hypertension, hypothyroidism, immune-related toxicity

★ CLINICAL PEARLS & EXAM FOCUS

▸ Any postmenopausal bleeding mandates endometrial sampling to exclude carcinoma – one of the most 'curable by early presentation' gynaecological cancers.

▸ Lynch syndrome screening (mismatch repair/microsatellite instability testing) should be routine in endometrial cancer, with implications for both treatment and family screening.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Uterine Neoplasms (current version)

M45 Epithelial Ovarian Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is ovarian cancer often diagnosed at an advanced stage? [Paper 2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise non-specific presenting symptoms and manage malignant ascites (CD4.1, PD3)

DEFINITION Often called a 'silent' cancer as symptoms (bloating, early satiety, pelvic discomfort) are vague and non-specific, leading to diagnosis at an advanced stage (III-IV) in the majority; high-grade serous carcinoma is the commonest and most lethal histological subtype, frequently BRCA-associated.

Stage / Setting	Principal Treatment Approach
Early (Stage I), rare at diagnosis	Surgical staging (hysterectomy, bilateral salpingo-oophorectomy, omentectomy, lymphadenectomy) ± adjuvant chemotherapy
Advanced (Stage III-IV), majority at diagnosis	Cytoreductive surgery (primary or interval after neoadjuvant chemotherapy) combined with platinum-taxane chemotherapy
Maintenance/relapsed disease	PARP inhibitor maintenance (especially BRCA-mutated/HRD-positive) ± bevacizumab; further chemotherapy on relapse

COMPLICATIONS

Disease-related:

- Malignant ascites with abdominal distension and early satiety
- Bowel obstruction from diffuse peritoneal carcinomatosis, sometimes multifocal and recurrent
- Pleural effusion (often right-sided) from transdiaphragmatic spread
- Omental caking and diffuse peritoneal studding at diagnosis

Treatment-related:

- Bevacizumab-related GI perforation, particularly in the setting of bowel involvement by tumour
- Surgical morbidity (anastomotic leak, prolonged ileus) from extensive cytoreductive surgery
- PARP inhibitor-related cytopenias and, rarely, secondary leukaemia
- Recurrent, often refractory malignant ascites requiring repeated paracentesis or a peritoneal catheter

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Carboplatin + Paclitaxel	First-line standard chemotherapy	Carboplatin AUC 5-6 + paclitaxel 175 mg/m² every 3 weeks, 6 cycles	Myelosuppression, peripheral neuropathy, hypersensitivity reactions
Olaparib	Maintenance, BRCA-mutated/HRD-positive disease	300 mg orally twice daily	Myelosuppression, fatigue, rare secondary MDS/AML
Bevacizumab		15 mg/kg IV every 3 weeks	

Added to chemotherapy		Hypertension, bleeding, GI
and/or maintenance		perforation, impaired wound
		healing

★ CLINICAL PEARLS & EXAM FOCUS

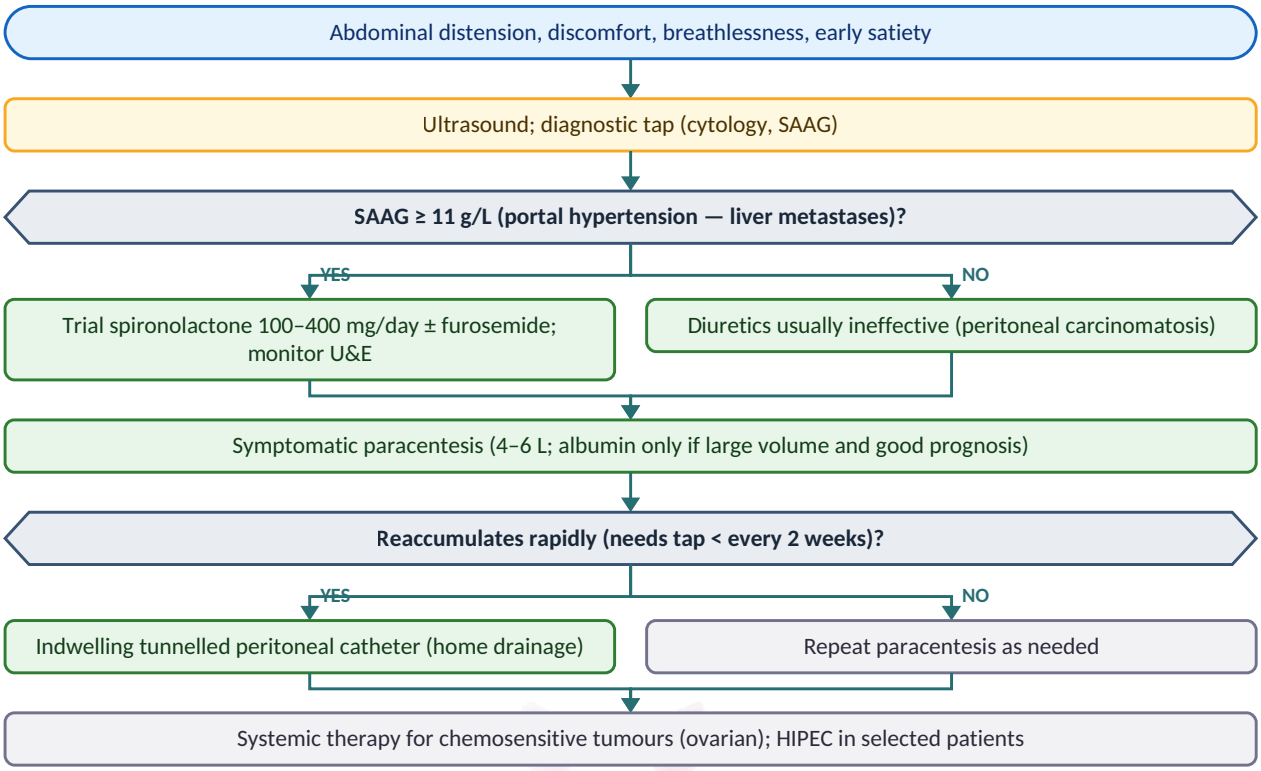
▸ BRCA/HRD testing should be performed in all high-grade serous ovarian cancer – it is central to maintenance therapy selection, not just family risk assessment.

▸ The 'silent' symptom profile (bloating, early satiety, urinary frequency) persisting beyond 2-3 weeks in a woman over 50 warrants prompt investigation.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Ovarian Cancer (current version)

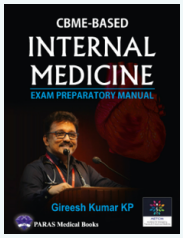
ALGORITHM · Malignant Ascites Algo 41 — full page ›



★ KEY POINTS

- ▸ Malignant ascites median survival is often < 6 months — discuss goals.
- ▸ Catumaxomab (EpCAM) was used in Europe for EpCAM+ ascites.

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M46 Vulvar Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How has sentinel lymph node biopsy changed the morbidity profile of vulvar cancer surgery? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Minimise surgical morbidity through sentinel node technique in gynaecological oncology (PD3, PD8)

DEFINITION Predominantly squamous cell carcinoma of the vulva, with two distinct pathways – HPV-related (younger women) and HPV-independent, often arising on a background of lichen sclerosus (older women); presents as a persistent vulvar lump, ulcer or pruritic lesion, often with diagnostic delay due to embarrassment.

Stage / Setting	Principal Treatment Approach
Early, localised (<2 cm, no suspicious nodes)	Wide local excision with sentinel lymph node biopsy to minimise morbidity
Locally advanced or node-positive	Radical vulvectomy with inguinofemoral lymphadenectomy, followed by adjuvant radiotherapy for high-risk features
Metastatic/unresectable	Platinum-based chemotherapy ± radiotherapy; limited evidence base given rarity

COMPLICATIONS

Disease-related:

- Inguinofemoral lymph node metastasis, the single most important prognostic factor
- Local destruction causing pain, bleeding, and secondary infection of the vulvar lesion
- Urethral/anal involvement from locally advanced disease
- Significant diagnostic delay from patient embarrassment, allowing progression

Treatment-related:

- Lymphoedema of the lower limbs after inguinofemoral lymphadenectomy, often chronic and distressing
- Wound breakdown/infection after radical vulvectomy, historically a major morbidity (reduced with sentinel node techniques)
- Psychosexual distress and altered body image after vulvar surgery
- Radiation-induced vulvar/groin skin desquamation

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation for locally advanced disease	40 mg/m² weekly during radiotherapy	Nephrotoxicity, myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

▸ Sentinel lymph node biopsy has substantially reduced lymphoedema and wound morbidity compared with historical complete inguinofemoral dissection, without compromising oncological outcomes in appropriately selected patients.

▸ Any persistent vulvar lesion, especially in a woman with known lichen sclerosus, should be biopsied rather than assumed benign.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Vulvar Cancer (current version)

M47 Vaginal Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why does the site of a vaginal tumour (upper vs lower third) matter for nodal staging? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply anatomical lymphatic drainage principles to gynaecological cancer staging (CD4.1)

DEFINITION A rare gynaecological malignancy, most often squamous cell carcinoma related to HPV, typically occurring in the upper posterior vaginal wall; historically also associated with in-utero diethylstilboestrol exposure causing clear-cell adenocarcinoma in younger women (now rare).

Stage / Setting	Principal Treatment Approach
Early, localised	Surgical resection (partial/radical vaginectomy) or definitive radiotherapy depending on site/extent
Locally advanced	Concurrent chemoradiation with brachytherapy boost, analogous to cervical cancer protocols
Metastatic	Platinum-based chemotherapy; limited specific trial evidence given rarity, often extrapolated from cervical cancer regimens

COMPLICATIONS

Disease-related:

- Local invasion into bladder (vesicovaginal fistula) or rectum (rectovaginal fistula) given the thin vaginal wall and close proximity to these structures
- Inguinal (lower third) or pelvic (upper two-thirds) lymph node metastasis depending on tumour location
- Vaginal stenosis complicating both disease and treatment
- Significant sexual dysfunction given anatomical site

Treatment-related:

- Vaginal stenosis and dyspareunia after radiotherapy, requiring vaginal dilator use to preserve function
- Fistula formation from locally advanced disease or treatment effect
- Radiation cystitis/proctitis given close proximity of bladder and rectum within treatment fields
- Profound psychosexual impact, often under-discussed by clinicians

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation	40 mg/m² weekly during radiotherapy	Nephrotoxicity, myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

▸ Vaginal dilator use should be proactively discussed and taught before radiotherapy-induced stenosis develops, not only after.

▸ Site within the vagina (upper vs lower third) determines lymphatic drainage pattern and therefore nodal staging fields – a useful anatomical teaching point.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Vaginal Cancer (current version)

M48 Gestational Trophoblastic Neoplasia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is gestational trophoblastic neoplasia considered uniquely curable even when metastatic? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Understand tumour-marker-guided treatment duration in a highly curable malignancy (CD2.1, CD4.1)

DEFINITION A spectrum of pregnancy-related tumours (invasive mole, choriocarcinoma, placental-site trophoblastic tumour) arising from trophoblastic tissue, uniquely trackable and highly curable using the tumour marker beta-hCG even in metastatic disease, distinguishing it from almost all other solid malignancies.

Stage / Setting	Principal Treatment Approach
Low-risk disease (FIGO/WHO scoring)	Single-agent methotrexate or actinomycin-D chemotherapy, with cure rates approaching 100%
High-risk disease	Combination chemotherapy (EMA-CO regimen) until beta-hCG normalises, plus consolidation cycles
Chemoresistant/relapsed disease	Salvage combination regimens; surgical resection of chemoresistant localised disease where feasible

COMPLICATIONS

Disease-related:

- Vaginal/uterine haemorrhage, sometimes massive, from highly vascular trophoblastic tissue
- Pulmonary metastases, the commonest metastatic site, occasionally causing haemoptysis
- Haemorrhagic brain metastases in high-risk/choriocarcinoma disease, a feared complication
- Uterine perforation from invasive mole

Treatment-related:

- Chemotherapy-related hepatotoxicity and myelosuppression requiring close monitoring during treatment to beta-hCG normalisation
- Secondary malignancy, a small but real long-term risk with etoposide-containing regimens
- Psychological distress related to both a cancer diagnosis and its origin in a pregnancy, often complicating grief/loss processing
- Future pregnancy counselling and monitoring needs after treatment completion

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose
Methotrexate	Low-risk disease, single-agent therapy	Weekly IM/ IV, per standard scoring-based protocol

CAUTION

Hepatotoxicity mucositis, myelosuppres – requires foli acid rescue in some protoco

EMA-CO (Etoposide, Methotrexate, Actinomycin-D, Cyclophosphamide, Vincristine)	High-risk disease	Per standard alternating weekly protocol

Myelosuppres alopecia, secondary malignancy ri with etoposid (rare)

★ CLINICAL PEARLS & EXAM FOCUS

► Beta-hCG is both diagnostic and a near-perfect tumour marker here – treatment continues until it normalises and for a defined consolidation period after, unlike marker-based monitoring in most other cancers.

► This is among the most curable metastatic malignancies in all of oncology – important for calibrated, accurate patient counselling despite the frightening 'choriocarcinoma' label.

GUIDELINES & RECENT ADVANCES

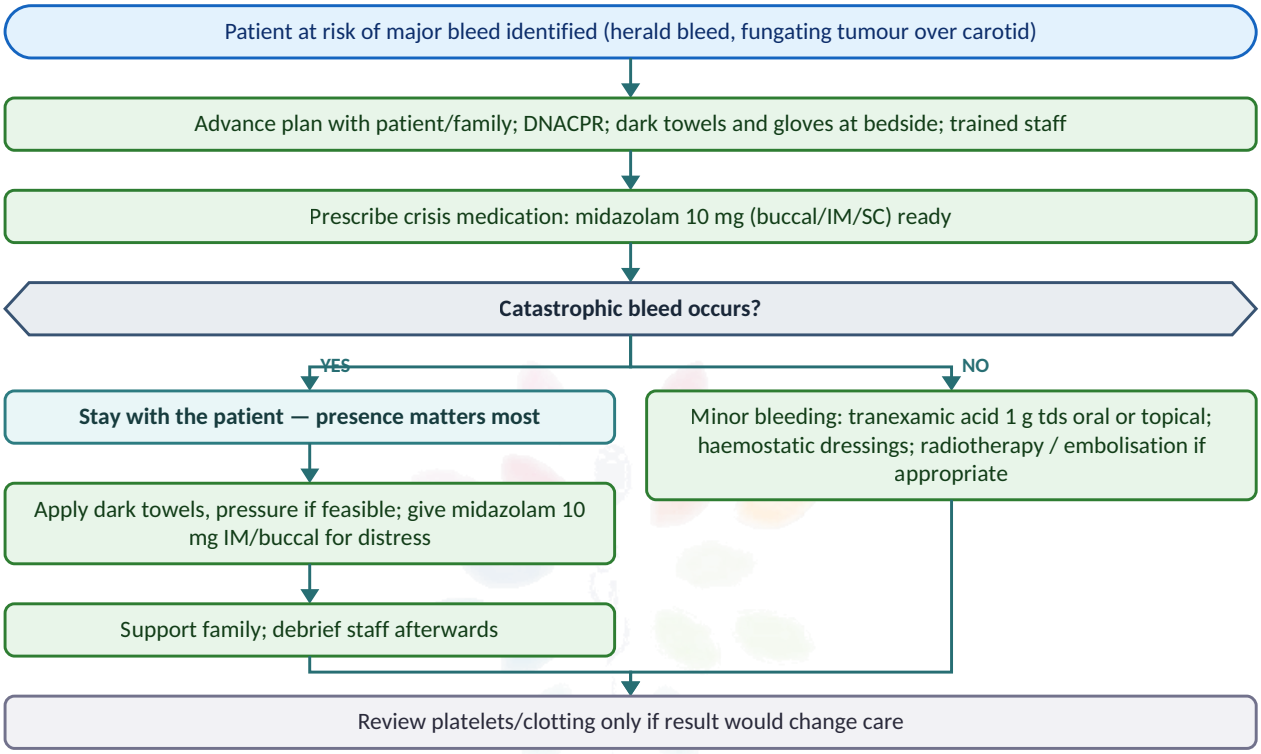
- FIGO/International Society for the Study of Trophoblastic Diseases Guidelines

EXAM-ORIENTED THEORY

Palliative care focus — gestational trophoblastic neoplasia

Issue	Approach
Highly curable even when metastatic	Palliative care runs alongside curative chemotherapy (EMA-CO)
Haemorrhage from vaginal / lung / brain metastases	Embolisation, tranexamic acid, radiotherapy
Psychological impact	Pregnancy loss, fertility anxiety — counselling
Follow-up	hCG monitoring; contraception during surveillance

ALGORITHM · Catastrophic Haemorrhage at the End of Life Algo 20 — full page ›



★ KEY POINTS

► Death from catastrophic bleed is usually within minutes — sedation may not take effect; calm presence is the key intervention.

► Use IM route if no IV access — SC absorption is unreliable in shock.

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M49 Fallopian Tube / Primary Peritoneal Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is the significance of the fallopian tube as a site of origin for 'ovarian' high-grade serous carcinoma? [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply current understanding of high-grade serous carcinogenesis to risk-reduction counselling (CD9, AD5)

DEFINITION High-grade serous carcinomas now understood to frequently originate in the fimbrial end of the fallopian tube (including in BRCA mutation carriers), clinically and therapeutically managed identically to epithelial ovarian cancer given shared biology and presentation.

Stage / Setting	Principal Treatment Approach
Early, localised (rare at diagnosis)	Surgical staging (as for ovarian cancer) ± adjuvant chemotherapy
Advanced (majority at diagnosis)	Cytoreductive surgery with platinum-taxane chemotherapy, identical to advanced ovarian cancer protocols
Maintenance/relapsed disease	PARP inhibitor maintenance (particularly BRCA-mutated) ± bevacizumab, as per ovarian cancer

COMPLICATIONS

Disease-related:

- Peritoneal carcinomatosis and malignant ascites, identical pattern to ovarian cancer
- Bowel obstruction from diffuse peritoneal disease
- Pleural effusion from transdiaphragmatic spread
- Often diagnosed incidentally during risk-reducing salpingo-oophorectomy in BRCA carriers (occult fallopian tube carcinoma)

Treatment-related:

- Identical surgical and chemotherapy-related complications to ovarian cancer (anastomotic leak, bevacizumab-related GI perforation, PARP inhibitor cytopenias)
- Psychological impact of an occult cancer diagnosis found during prophylactic surgery in BRCA carriers
- Recurrent malignant ascites requiring repeated drainage

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Carboplatin + Paclitaxel	First-line chemotherapy	Carboplatin AUC 5-6 + paclitaxel 175 mg/m² every 3 weeks	Myelosuppression, peripheral neuropathy
Olaparib	Maintenance, BRCA-mutated disease	300 mg orally twice daily	Myelosuppression, fatigue

★ CLINICAL PEARLS & EXAM FOCUS

► This entity underlies the rationale for salpingectomy (removing the fallopian tubes, the presumed site of origin) as an interim risk-reduction strategy in BRCA carriers not yet ready for oophorectomy.

► Management mirrors ovarian cancer completely – the distinction matters more for understanding disease origin/screening than for altering treatment.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Ovarian Cancer (includes Fallopian Tube and Primary Peritoneal Cancer)

M50 Acute Myeloid Leukaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is empirical antibiotic therapy started immediately in febrile neutropenia? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage febrile neutropenia as an oncological emergency (CD4.1, PD3)

DEFINITION An aggressive clonal proliferation of myeloid blasts in the bone marrow, presenting with cytopenia-related symptoms (fatigue, infection, bleeding) over days to weeks; cytogenetic/molecular profiling (FLT3, NPM1, core-binding factor abnormalities) is central to risk stratification and treatment choice.

Stage / Setting	Principal Treatment Approach
Fit, favourable/intermediate risk	Intensive induction chemotherapy (7+3: cytarabine-anthracycline) followed by consolidation ± FLT3 inhibitor if FLT3-mutated
Fit, adverse risk	Induction chemotherapy followed by allogeneic stem cell transplantation in first remission
Unfit for intensive therapy (elderly/comorbid)	Hypomethylating agent (azacitidine) combined with venetoclax, a lower-intensity but effective approach

COMPLICATIONS

Disease-related:

- Febrile neutropenia and life-threatening sepsis during the profound cytopenic phase
- Disseminated intravascular coagulation, particularly in acute promyelocytic leukaemia (a related but distinct, highly curable subtype)
- Leukostasis in hyperleukocytosis causing pulmonary/CNS complications
- Tumour lysis syndrome with rapid cytoreduction

Treatment-related:

- Prolonged pancytopenia requiring transfusion support and strict infection precautions
- Differentiation syndrome if acute promyelocytic leukaemia is present and treated with ATRA/arsenic
- Graft-versus-host disease after allogeneic transplantation
- Cardiotoxicity from cumulative anthracycline exposure

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cytarabine + Daunorubicin (7+3)	Intensive induction, fit patients	Cytarabine 100-200 mg/m²/ day x7 days + daunorubicin 60-90 mg/m²/day x3 days	Profound myelosuppression, cardiotoxicity, severe mucositis, tumour lysis syndrome
Azacitidine + Venetoclax	Unfit/elderly patients	Azacitidine 75 mg/m² days 1-7 + venetoclax 400 mg daily (dose-ramped), 28-day cycles	Myelosuppression, tumour lysis syndrome at initiation, GI upset

★ CLINICAL PEARLS & EXAM FOCUS

► Suspected acute promyelocytic leukaemia (characteristic Auer rods, DIC) should prompt immediate ATRA initiation even before genetic confirmation, given the high risk of fatal early haemorrhage.

► Febrile neutropenia in AML is a medical emergency requiring empirical broad-spectrum antibiotics within one hour of presentation.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Acute Myeloid Leukemia (current version)

M51 Acute Lymphoblastic Leukaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is CNS-directed prophylaxis essential in ALL even without CNS symptoms? [Paper 1, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Understand sanctuary-site pharmacology in haematological malignancy treatment (CD2.1, CD4.1)

DEFINITION A malignancy of lymphoid precursor cells, predominantly a childhood cancer (though it also occurs in adults with a worse prognosis), presenting with marrow failure, lymphadenopathy, hepatosplenomegaly and sometimes CNS involvement at diagnosis.

Stage / Setting	Principal Treatment Approach
Paediatric, standard risk	Multi-phase chemotherapy (induction, consolidation, maintenance) with CNS-directed therapy (intrathecal chemotherapy), achieving cure rates over 90%
Adult/high-risk, Philadelphia chromosome-negative	Intensive paediatric-inspired multi-agent chemotherapy protocols
Philadelphia chromosome-positive	Tyrosine kinase inhibitor (imatinib/dasatinib) combined with chemotherapy, often followed by allogeneic transplantation

COMPLICATIONS

Disease-related:

- CNS leukaemic infiltration causing headache, cranial nerve palsies or meningism
- Mediastinal mass (T-cell ALL) causing airway/SVC compression
- Tumour lysis syndrome with rapid cytoreduction given chemosensitivity
- Testicular relapse, a recognised sanctuary site in boys

Treatment-related:

- Asparaginase-related pancreatitis, hepatotoxicity, and thrombosis (including cerebral venous thrombosis)
- Vincristine-induced peripheral neuropathy and constipation
- Steroid-related hyperglycaemia, mood disturbance, and avascular necrosis with prolonged courses
- Graft-versus-host disease after allogeneic transplantation in high-risk/relapsed disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Vincristine + Prednisolone + Asparaginase	Induction chemotherapy backbone	Per standard protocol-specific dosing and schedule	Vincristine neuropathy, asparaginase pancreatitis/ thrombosis, steroid hyperglycaemia
Imatinib	Philadelphia chromosome-positive disease, added to chemotherapy	400-600 mg orally once daily	Fluid retention, cytopenias, hepatotoxicity

★ CLINICAL PEARLS & EXAM FOCUS

▸ Mediastinal mass with airway/SVC compromise at presentation of T-cell ALL is an emergency requiring urgent, often empirical steroid therapy before definitive diagnosis.

▸ CNS-directed prophylaxis (intrathecal chemotherapy ± cranial irradiation) is essential even without overt CNS disease, given the CNS as a pharmacological sanctuary site.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Acute Lymphoblastic Leukemia (current version)

M52 Chronic Myeloid Leukaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How has tyrosine kinase inhibitor therapy changed the natural history of CML? [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Understand molecular monitoring and treatment adherence in chronic oral oncology therapy (CD2.1, PD2)

DEFINITION A myeloproliferative neoplasm defined by the BCR-ABL1 fusion gene (Philadelphia chromosome), often discovered incidentally on a routine blood count showing marked leucocytosis; the advent of tyrosine kinase inhibitors has transformed this from a fatal disease into a chronic, manageable condition.

Stage / Setting	Principal Treatment Approach
Chronic phase	Oral tyrosine kinase inhibitor (imatinib, or a second-generation agent for higher-risk disease) as first-line, continued indefinitely or until treatment-free remission criteria are met
Accelerated/blast phase	Higher-potency TKI ± chemotherapy, with allogeneic transplantation considered for blast phase or TKI-resistant disease
TKI-resistant/ intolerant disease	Switch to an alternative TKI based on resistance mutation profile (e.g. ponatinib for T315I mutation)

COMPLICATIONS

Disease-related:

- Massive splenomegaly causing early satiety and left upper quadrant discomfort at diagnosis
- Leukostasis in markedly elevated white cell counts causing visual disturbance or priapism
- Progression to blast crisis (acute leukaemia transformation) if untreated or resistant
- Hyperviscosity-related thrombotic events

Treatment-related:

- TKI-related pleural effusion (dasatinib) or arterial occlusive events (ponatinib)
- Cytopenias limiting TKI dose intensity, particularly early in treatment
- Treatment non-adherence leading to loss of molecular response – a major real-world complication given lifelong oral therapy
- Graft-versus-host disease if transplantation is required for resistant/advanced disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Imatinib	First-line chronic phase	400 mg orally once daily	Fluid retention, periorbital oedema, cytopenias, GI upset
Dasatinib	First- or second-line, higher-risk disease or imatinib resistance	100 mg orally once daily	Pleural effusion, pulmonary hypertension (rare), cytopenias
Ponatinib			
	T315I-mutated or multi-TKI-resistant disease	45 mg orally once daily (dose-reduced per response/tolerability)	Arterial thrombosis/occlusion – requires cardiovascular risk assessment

★ CLINICAL PEARLS & EXAM FOCUS

▸ Regular molecular monitoring (BCR-ABL1 transcript levels by PCR) guides treatment decisions and is central to the modern management paradigm.

▸ Treatment adherence counselling is critical – CML has become a model of a cancer managed as a chronic disease with a once-daily oral drug, but only if taken consistently.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Chronic Myeloid Leukemia (current version)

M53 Chronic Lymphocytic Leukaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is 'watch and wait' appropriate in early-stage CLL? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply evidence-based observation strategies in indolent haematological malignancy (CD4.1, AD3)

DEFINITION The commonest adult leukaemia in Western populations, a clonal proliferation of mature B-lymphocytes, often indolent and discovered incidentally on a routine blood count showing lymphocytosis; many patients never require treatment.

Stage / Setting	Principal Treatment Approach
Early-stage, asymptomatic	Watch and wait – active surveillance without treatment, as early intervention does not improve survival
Symptomatic/progressive disease	BTK inhibitor (ibrutinib/acalabrutinib) or venetoclax-obinutuzumab as first-line targeted therapy
Relapsed/refractory or high-risk cytogenetics (del17p/TP53 mutated)	Alternative targeted agent class switch (BTK inhibitor to venetoclax-based regimen or vice versa)

COMPLICATIONS

Disease-related:

- Recurrent infections from hypogammaglobulinaemia and immune dysfunction, a major cause of morbidity/mortality
- Autoimmune haemolytic anaemia or immune thrombocytopenia (autoimmune cytopenias)
- Richter transformation – progression to an aggressive lymphoma (usually DLBCL), signalled by rapid lymph node growth or B symptoms
- Massive lymphadenopathy/splenomegaly causing mechanical symptoms

Treatment-related:

- Tumour lysis syndrome with venetoclax initiation, requiring a carefully staged dose ramp-up and monitoring
- Atrial fibrillation and bleeding risk with BTK inhibitors, requiring caution with concurrent anticoagulation
- Increased infection risk from both disease-related immune dysfunction and treatment-related immunosuppression
- Secondary malignancies, including skin cancers, more common in CLL patients generally

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Ibrutinib	First-line or relapsed disease	420 mg orally once daily	Atrial fibrillation, bleeding risk, hypertension, infection risk
Venetoclax + Obinutuzumab	First-line, time-limited treatment approach	Venetoclax dose-ramped to 400 mg daily + obinutuzumab per standard schedule	Tumour lysis syndrome at initiation (requires risk-stratified ramp-up), neutropenia

★ CLINICAL PEARLS & EXAM FOCUS

▸ 'Watch and wait' is an evidence-based, not a passive or inadequate, strategy in early asymptomatic CLL – important for patient reassurance and counselling.

▸ Recurrent infections are often the major clinical burden in CLL, sometimes more so than the leukaemia itself – prophylactic immunoglobulin replacement is considered in selected cases.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma (current version)

M54 Hairy Cell Leukaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why does hairy cell leukaemia characteristically give a 'dry tap' on bone marrow aspiration? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Correlate marrow pathology with diagnostic technique in haematological malignancy (CD2.1)

DEFINITION A rare, indolent B-cell leukaemia characterised by cells with distinctive cytoplasmic projections ('hairy cells'), presenting with pancytopenia, massive splenomegaly, and a characteristic 'dry tap' on bone marrow aspiration due to marrow fibrosis.

Stage / Setting	Principal Treatment Approach
Symptomatic disease (cytopenias, symptomatic splenomegaly)	Purine analogue chemotherapy (cladribine) as first-line, achieving durable remission in the majority
Relapsed disease	Repeat purine analogue course, or rituximab-based combination therapy
Refractory disease	BRAF inhibitor (vemurafenib, given near-universal BRAF V600E mutation) ± rituximab

COMPLICATIONS

Disease-related:

- Recurrent infections (including atypical/opportunistic) from profound monocytopenia and immune dysfunction
- Massive splenomegaly causing abdominal discomfort and hypersplenism-related cytopenia
- Bone marrow fibrosis causing the classic 'dry tap' and diagnostic difficulty
- Vasculitic skin complications, occasionally seen in this disease

Treatment-related:

- Prolonged immunosuppression after cladribine therapy, with increased opportunistic infection risk for months
- Secondary cutaneous malignancies with BRAF inhibitor therapy
- Tumour lysis syndrome, uncommon but possible with rapid treatment response
- Splenic rupture, a rare but serious complication of massive splenomegaly

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cladribine	First-line and relapsed disease	0.1 mg/kg/day continuous IV infusion for 7 days (single course)	Profound, prolonged myelosuppression and immunosuppression, increased infection risk
Vemurafenib	Refractory/relapsed disease, BRAF-mutated	960 mg orally twice daily	Photosensitivity, arthralgia, secondary cutaneous squamous cell carcinoma

★ CLINICAL PEARLS & EXAM FOCUS

▸ A single course of cladribine achieves durable remission in most patients – important to counsel that this is a highly treatable, often curable-in-practice disease despite its rarity.

▸ Nearly universal BRAF V600E mutation makes vemurafenib a reliable option in refractory disease – a good recent-advances teaching point.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Hairy Cell Leukemia (current version)

M55 Hodgkin Lymphoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss response-adapted therapy and its role in minimising long-term toxicity in Hodgkin lymphoma. [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Balance cure intent with long-term survivorship risk in young oncology patients (AD5, CD4.1)

DEFINITION A B-cell lymphoma characterised by the pathognomonic Reed-Sternberg cell within a background of reactive inflammatory cells, typically presenting with painless cervical/supraclavicular lymphadenopathy and sometimes B symptoms (fever, night sweats, weight loss); highly curable even at advanced stage.

Stage / Setting	Principal Treatment Approach
Early-stage, favourable	Combined modality therapy – abbreviated ABVD chemotherapy followed by involved-site radiotherapy
Early-stage, unfavourable or advanced-stage	Full-course ABVD (or escalated BEACOPP for high-risk advanced disease) ± consolidation radiotherapy
Relapsed/refractory disease	Salvage chemotherapy followed by high-dose chemotherapy with autologous stem cell transplantation; brentuximab vedotin or checkpoint inhibitors for further relapse

COMPLICATIONS

Disease-related:

- Mediastinal bulky disease causing airway or superior vena cava compression
- B symptoms (fever, drenching night sweats, >10% weight loss) reflecting systemic cytokine-driven disease
- Pruritus, sometimes severe and a presenting symptom in its own right
- Contiguous nodal spread, historically the basis for Ann Arbor staging

Treatment-related:

- Bleomycin-induced pulmonary fibrosis, a serious and sometimes irreversible toxicity requiring pulmonary function monitoring
- Secondary malignancies (breast cancer, lung cancer, leukaemia) as late effects of chemotherapy/ radiotherapy, particularly relevant given young patient age at diagnosis
- Cardiotoxicity from anthracycline exposure, with long-term cardiac surveillance needed in survivors
- Infertility risk from alkylating-agent-containing regimens, relevant to fertility preservation counselling

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
ABVD (Doxorubicin, Bleomycin, Vinblastine, Dacarbazine)	Standard first-line chemotherapy	Standard 28-day cycle, 2-6 cycles depending	Bleomycin pulmonary toxicity, cardiotoxicity
		on stage/ response	(doxorubicin), myelosuppression
Brentuximab Vedotin	Relapsed/ refractory or in combination with AVD first-line for advanced disease	1.8 mg/kg IV every 3 weeks	Peripheral neuropathy, myelosuppression, infusion reactions

★ CLINICAL PEARLS & EXAM FOCUS

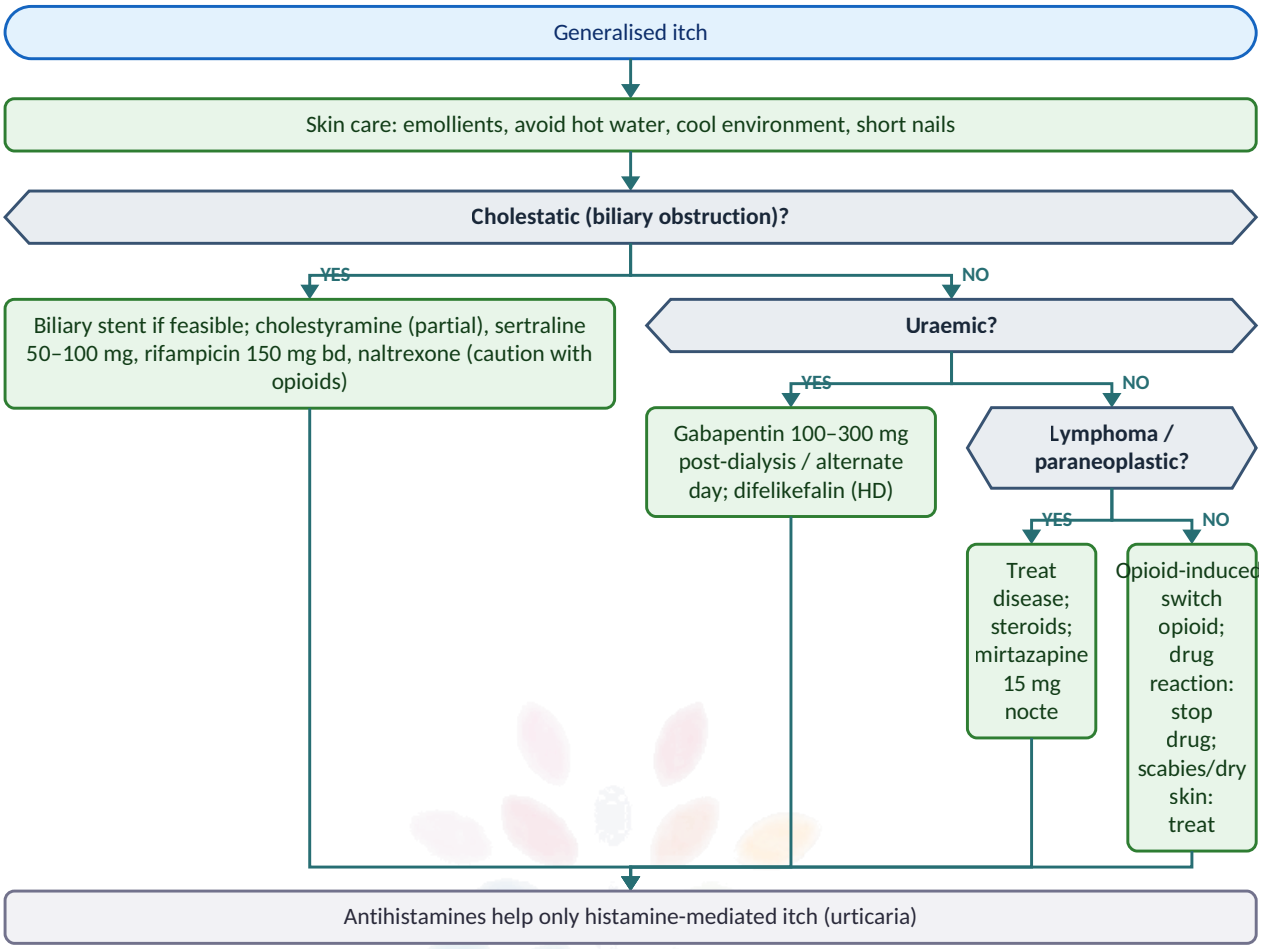
▸ Hodgkin lymphoma has one of the highest cure rates in oncology – balancing cure with minimising long-term treatment-related morbidity (cardiac, pulmonary, secondary malignancy) is the central modern management theme.

▸ PET-CT response-adapted therapy allows treatment de-escalation in good responders, reducing long-term toxicity without compromising cure rates – an important recent-advances point.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Hodgkin Lymphoma (current version)

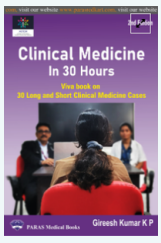
ALGORITHM · Pruritus in Palliative Care Algo 57 — full page ›



★ KEY POINTS

- ▸ Opioid antagonists can precipitate pain and withdrawal in patients on opioids.
- ▸ Sertraline and mirtazapine are useful in mixed causes.

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M56 Diffuse Large B-Cell Lymphoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is cytokine release syndrome and how is it managed in CAR-T therapy? [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Understand novel cellular therapy toxicities relevant to supportive/palliative oncology (CD2.1, PD4)

DEFINITION The commonest aggressive non-Hodgkin lymphoma, presenting with rapidly enlarging lymphadenopathy ± extranodal involvement (GI tract, bone, CNS) and B symptoms; despite aggressive behaviour, it is potentially curable with standard immunochemotherapy.

Stage / Setting	Principal Treatment Approach
Limited stage	Abbreviated R-CHOP chemoimmunotherapy (3-4 cycles) with involved-site radiotherapy, or full-course R-CHOP alone
Advanced stage	Full-course R-CHOP (6-8 cycles) ± CNS prophylaxis in high-risk sites (testis, paranasal sinus, high IPI score)
Relapsed/refractory disease	Salvage chemotherapy with autologous stem cell transplantation, or CAR-T cell therapy for transplant-ineligible/further relapse

COMPLICATIONS

Disease-related:

- Tumour lysis syndrome at treatment initiation given rapid chemosensitivity, particularly in bulky disease
- CNS relapse, particularly in high-risk extranodal sites, often with poor prognosis once it occurs
- Spinal cord compression from epidural extension in vertebral involvement
- GI perforation/bleeding from rapid tumour lysis at a GI primary site during treatment

Treatment-related:

- Cytokine release syndrome and neurotoxicity (ICANS) with CAR-T cell therapy, requiring specialised inpatient monitoring and tocilizumab/steroid protocols
- Cardiotoxicity from cumulative anthracycline exposure
- Febrile neutropenia during R-CHOP cycles
- Secondary CNS lymphoma, sometimes despite prophylaxis in very high-risk cases

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose
R-CHOP (Rituximab, Cyclophosphamide, Doxorubicin, Vincristine, Prednisolone)	Standard first-line therapy	Standard 21-day cycle, 6-8 cycles

CAUTION

Myelosupp cardiotoxic (doxorubici peripheral neuropathy (vincristine tumour lysi

CAR-T Cell Therapy (e.g. axicabtagene ciloleucel)	Relapsed/ refractory disease after ≥2 prior lines	Single infusion after lymphodepleting chemotherapy

syndrome a initiation

Cytokine re syndrome, neurotoxici (ICANS) – re specialised monitoring

★ CLINICAL PEARLS & EXAM FOCUS

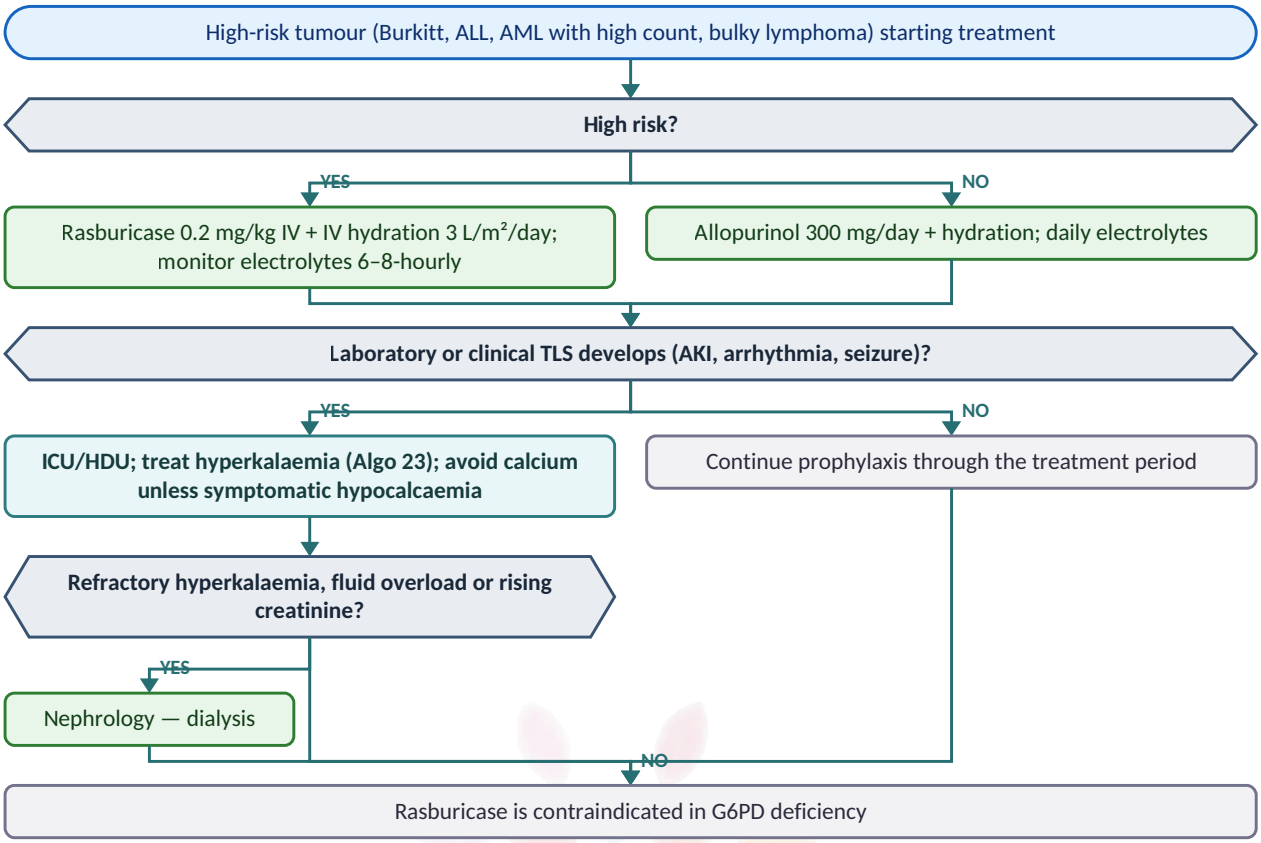
► Pre-emptive tumour lysis syndrome prophylaxis (hydration, allopurinol/rasburicase) is essential before starting chemotherapy in bulky DLBCL.

► CAR-T cell therapy has transformed outcomes in relapsed/refractory disease but requires specialised centres equipped to manage cytokine release syndrome – an important recent-advances/systems point.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: B-Cell Lymphomas (current version)

ALGORITHM · Tumour Lysis Syndrome Algo 18 — full page ›



★ KEY POINTS

- ▸ Cairo–Bishop criteria define laboratory and clinical TLS.
- ▸ Do not alkalinise urine routinely — calcium phosphate may precipitate.

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M57 Follicular Lymphoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must hepatitis B status be checked before starting rituximab? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Screen for and prevent treatment-related viral reactivation in immunosuppressive oncology therapy (CD4.1, PD9)

DEFINITION The commonest indolent non-Hodgkin lymphoma, characterised by the t(14;18) translocation and BCL2 overexpression, typically presenting with slowly progressive, painless lymphadenopathy; generally not curable with standard therapy but compatible with long survival, sometimes managed expectantly.

Stage / Setting	Principal Treatment Approach
Asymptomatic, low tumour burden	Watch and wait, as early treatment does not improve overall survival in this setting
Symptomatic/high tumour burden	Rituximab-based chemoimmunotherapy (R-CHOP or bendamustine-rituximab) followed by rituximab maintenance
Relapsed/transformed disease	Alternative chemoimmunotherapy regimen, or treatment as per transformed aggressive lymphoma if transformation to DLBCL occurs

COMPLICATIONS

Disease-related:

- Histological transformation to an aggressive lymphoma (usually DLBCL), signalled by rapid nodal growth, new B symptoms or disproportionate LDH rise
- Bulky lymphadenopathy causing mechanical compression of adjacent structures
- Recurrent, smouldering disease course over many years with repeated need for treatment
- Bone marrow involvement causing cytopenias

Treatment-related:

- Hepatitis B reactivation with rituximab-containing therapy – screening and prophylaxis is mandatory before starting treatment
- Prolonged immunosuppression and infection risk from extended rituximab maintenance
- Myelosuppression from bendamustine, occasionally prolonged
- Progressive disease of treatment (PDT) within 24 months of initial therapy, identifying a higher-risk subgroup

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Rituximab + Bendamustine	First-line symptomatic disease	Rituximab 375 mg/m² + bendamustine 90 mg/m² days 1-2, every 28 days	Myelosuppression, infusion reactions, opportunistic infection risk
Rituximab	Maintenance therapy after induction	375 mg/m² IV every 2 months for up to 2 years	Infusion reactions, prolonged B-cell depletion/infection risk, hepatitis B reactivation risk

★ CLINICAL PEARLS & EXAM FOCUS

▸ Hepatitis B screening before rituximab is mandatory and frequently tested – reactivation can be fatal if missed.

▸ Watch and wait remains appropriate in asymptomatic, low-burden disease, similar in principle to CLL, despite patient anxiety about 'untreated cancer'.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: B-Cell Lymphomas (current version)

M58 Mantle Cell Lymphoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the typical pattern of extranodal involvement in mantle cell lymphoma. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise aggressive lymphoma variants requiring intensified treatment approaches (CD2.1, CD4.1)

DEFINITION An aggressive-behaving but biologically intermediate B-cell lymphoma defined by the t(11;14) translocation and cyclin D1 overexpression, often presenting with advanced-stage disease at diagnosis including bone marrow and GI tract (lymphomatous polyposis) involvement.

Stage / Setting	Principal Treatment Approach
Fit patients	Intensive chemoimmunotherapy (e.g. R-DHAP alternating with R-CHOP, or similar intensive regimens) followed by autologous stem cell transplantation and rituximab maintenance
Less fit/elderly patients	Less intensive chemoimmunotherapy (bendamustine-rituximab) followed by rituximab maintenance
Relapsed/ refractory disease	BTK inhibitor (ibrutinib/acalabrutinib) or CAR-T cell therapy for further relapse

COMPLICATIONS

Disease-related:

- Diffuse GI tract involvement (lymphomatous polyposis) causing bleeding, obstruction or perforation
- Extensive bone marrow infiltration causing cytopenias at diagnosis
- CNS involvement in a subset, particularly at relapse
- Rapid clinical progression despite initially appearing indolent in some cases (blastoid variant particularly aggressive)

Treatment-related:

- Severe, prolonged myelosuppression from intensive induction regimens
- Cardiac arrhythmia (atrial fibrillation) with BTK inhibitor therapy
- Graft-versus-host disease or relapse after autologous/allogeneic transplantation
- Cytokine release syndrome with CAR-T cell therapy in relapsed disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Bendamustine + Rituximab	First-line, less intensive approach	Bendamustine 90 mg/m² days 1-2 + rituximab 375 mg/m², every 28 days	Myelosuppression, infusion reactions
Ibrutinib	Relapsed/refractory disease	560 mg orally once daily	Atrial fibrillation, bleeding risk, infection

★ CLINICAL PEARLS & EXAM FOCUS

► Mantle cell lymphoma often presents with advanced, marrow- and GI-involved disease despite patients feeling comparatively well – staging should be thorough even with limited symptoms.

► The blastoid variant behaves considerably more aggressively and should prompt more intensive treatment consideration.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: B-Cell Lymphomas (current version)

M59 Burkitt Lymphoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is Burkitt lymphoma treated as an oncological emergency? [Paper 2, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and pre-emptively manage extreme tumour lysis syndrome risk (CD4.1, PD3)

DEFINITION The most rapidly proliferating human malignancy, driven by MYC gene translocation, occurring in endemic (EBV-associated, jaw/facial, African children), sporadic (abdominal, often ileocaecal) and immunodeficiency-associated (HIV-related) forms; requires emergency-level treatment initiation given its explosive growth rate.

Stage / Setting	Principal Treatment Approach
All stages	Intensive, short-duration, dose-dense combination chemotherapy (e.g. CODOX-M/IVAC or hyper-CVAD) with mandatory CNS-directed therapy, started urgently
HIV-associated disease	Same intensive chemotherapy backbone combined with concurrent antiretroviral therapy
CNS involvement at diagnosis	Intensified CNS-directed chemotherapy (high-dose methotrexate/cytarabine, intrathecal therapy)

COMPLICATIONS

Disease-related:

- Severe tumour lysis syndrome, almost universal risk given the extremely high proliferation rate – can occur even before chemotherapy from spontaneous cell turnover
- Bowel obstruction/intussusception from ileocaecal (sporadic form) involvement
- Jaw/facial bone destruction in endemic (African) form
- CNS involvement, common and requiring aggressive prophylaxis/treatment

Treatment-related:

- Acute kidney injury from tumour lysis syndrome despite prophylaxis
- Profound myelosuppression and mucositis from dose-dense chemotherapy
- Bowel perforation if chemotherapy rapidly lyses a bulky abdominal mass adherent to bowel wall
- Opportunistic infection risk, particularly in HIV-associated disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
CODOX-M/ IVAC	Standard intensive chemotherapy regimen	Per alternating standard protocol cycles with aggressive supportive care	Severe myelosuppression, mucositis, tumour lysis syndrome – among the highest risk of any regimen
Rasburicase	Tumour lysis syndrome prophylaxis, mandatory given extreme risk	0.2 mg/kg IV once daily for up to 5-7 days as needed	Haemolysis in G6PD deficiency – screen before use

★ CLINICAL PEARLS & EXAM FOCUS

► Burkitt lymphoma is a true oncological emergency – treatment must begin within 24-48 hours of diagnosis, with tumour lysis prophylaxis started even before the first chemotherapy dose.

► G6PD screening is required before rasburicase, as it can precipitate severe haemolysis in deficient patients.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: B-Cell Lymphomas (current version)

M60 Mycosis Fungoides / Sezary Syndrome

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the management of severe pruritus in cutaneous T-cell lymphoma. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage refractory pruritus and skin-barrier complications in cutaneous lymphoma (PD3, CD6)

DEFINITION The commonest cutaneous T-cell lymphoma, presenting with slowly evolving patches, plaques and eventually tumours of the skin over years; Sezary syndrome is the leukaemic, erythrodermic variant with circulating malignant T-cells and a worse prognosis.

Stage / Setting	Principal Treatment Approach
Early-stage, patch/plaque (skin-limited)	Skin-directed therapy – topical corticosteroids, phototherapy (PUVA/UVB), or topical nitrogen mustard
Advanced skin disease/tumours	Total skin electron beam therapy or systemic retinoids/HDAC inhibitors added to skin-directed therapy
Sezary syndrome/advanced systemic disease	Extracorporeal photopheresis, systemic chemotherapy, mogamulizumab, or allogeneic transplantation in eligible patients

COMPLICATIONS

Disease-related:

- Erythroderma causing severe pruritus, thermoregulatory dysfunction and secondary skin infection
- Recurrent skin infections (Staphylococcus aureus colonisation/superinfection) from disrupted skin barrier
- Progression to systemic/visceral involvement (lymph nodes, blood, viscera) in advanced disease
- Severe, often treatment-refractory pruritus significantly impairing quality of life

Treatment-related:

- Hypertriglyceridaemia (sometimes severe, pancreatitis risk) with bexarotene therapy
- Central hypothyroidism with bexarotene, requiring thyroid hormone supplementation
- Secondary skin infections complicating disrupted skin barrier function
- Graft-versus-host disease after allogeneic transplantation in advanced disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Bexarotene	Systemic retinoid therapy for refractory/advanced disease	300 mg/m²/day orally	Hypertriglyceridaemia (can be severe), central hypothyroidism, hepatotoxicity
Mogamulizumab	Relapsed/refractory mycosis fungoides/Sezary syndrome	1 mg/kg IV weekly for 4 weeks then every 2 weeks	Infusion reactions, skin rash (can mimic disease), autoimmune complications

★ CLINICAL PEARLS & EXAM FOCUS

▸ Pruritus in cutaneous T-cell lymphoma is often severe and disproportionate to visible skin findings – proactive, multimodal symptom management is essential, not an afterthought.

▸ Erythrodermic patients are at risk of high-output cardiac failure and temperature dysregulation – monitor fluid status and temperature closely in Sezary syndrome.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Primary Cutaneous Lymphomas (current version)

M61 Multiple Myeloma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the CRAB criteria and their relevance to initiating treatment in myeloma. [Paper 1, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage bone disease, renal impairment and infection risk in plasma cell malignancy (CD3.1, CD4.1, PD3)

DEFINITION A clonal plasma cell malignancy producing monoclonal immunoglobulin (M protein), classically presenting with the CRAB features (hyperCalcaemia, Renal impairment, Anaemia, Bone lesions); a highly relevant malignancy for palliative medicine given its bone pain burden and renal/infectious complications.

Stage / Setting	Principal Treatment Approach
Transplant-eligible	Induction triplet therapy (bortezomib-lenalidomide-dexamethasone) followed by autologous stem cell transplantation and maintenance lenalidomide
Transplant-ineligible	Induction triplet/quadruplet therapy continued without transplantation, followed by maintenance therapy
Relapsed/refractory disease	Alternative-class combination (daratumumab-based regimens, or newer agents such as CAR-T/ bispecific antibodies) depending on prior therapy

COMPLICATIONS

Disease-related:

- Pathological fracture and severe bone pain from lytic bone lesions, often the presenting complaint
- Renal failure from light-chain cast nephropathy, hypercalcaemia or amyloid deposition
- Recurrent infections from functional hypogammaglobulinaemia and immune dysfunction
- Hyperviscosity syndrome in high M-protein states causing visual disturbance, bleeding, neurological symptoms

Treatment-related:

- Osteonecrosis of the jaw from long-term bisphosphonate/denosumab therapy
- Peripheral neuropathy from bortezomib, sometimes dose-limiting
- Thalidomide/lenalidomide-associated venous thromboembolism, requiring prophylaxis
- Secondary infections given both disease- and treatment-related immunosuppression – a leading cause of myeloma mortality

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Bortezomib + Lenalidomide + Dexamethasone (VRd)	Standard first-line induction	Per standard 21/28-day protocol	Peripheral neuropathy (bortezomib), thrombosis risk (lenalidomide, requires prophylactic anticoagulation/aspirin),
			hyperglycaemia (dexamethasone)
Zoledronic Acid	Bone disease, started at diagnosis regardless of imaging findings	4 mg IV every 4 weeks (renally adjusted)	Osteonecrosis of jaw, renal impairment – dental assessment and renal monitoring required
Daratumumab	Relapsed/ refractory or added to first-line regimens	16 mg/kg IV per approved schedule, or subcutaneous formulation	Infusion reactions (IV), increased infection risk, interference with blood typing/ crossmatch

★ CLINICAL PEARLS & EXAM FOCUS

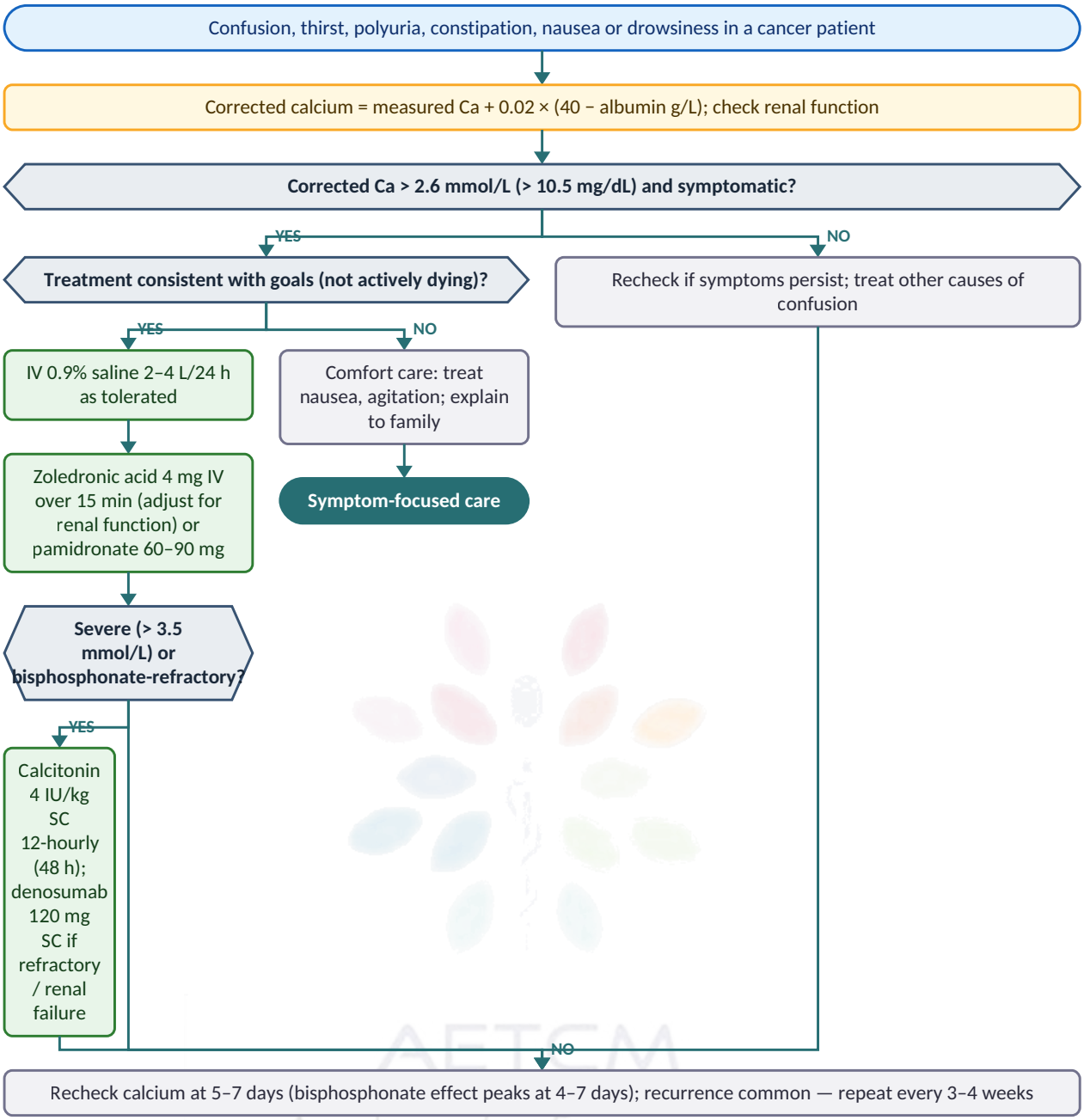
► Bone-targeted therapy (bisphosphonate or denosumab) should be started at diagnosis in myeloma regardless of whether lytic lesions are seen on initial imaging, given the near-universal bone disease risk.

► Infection is a leading cause of death in myeloma – vaccination (pneumococcal, influenza) and prompt treatment of febrile episodes are essential palliative/supportive measures.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Multiple Myeloma (current version)

ALGORITHM · Approach to Hypercalcaemia of Malignancy Algo 10 — full page ›



★ KEY POINTS

- ▸ Median survival after cancer hypercalcaemia is often weeks to months — discuss goals.
- ▸ PTHrP-mediated humoral hypercalcaemia is commonest (squamous carcinomas).

M62 Waldenstrom Macroglobulinaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why might rituximab transiently worsen hyperviscosity symptoms, and how is this managed? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise and urgently manage hyperviscosity syndrome (CD4.1, PD3)

DEFINITION A rare lymphoplasmacytic lymphoma characterised by monoclonal IgM production, often causing hyperviscosity syndrome given IgM's large pentameric size; associated with MYD88 L265P mutation in the majority of cases.

Stage / Setting	Principal Treatment Approach
Asymptomatic disease	Watch and wait, as for other indolent lymphomas, given lack of survival benefit from early treatment
Symptomatic disease (anaemia, hyperviscosity, bulky disease)	BTK inhibitor (ibrutinib/zanubrutinib) or rituximab-based chemoimmunotherapy (bendamustine-rituximab)
Hyperviscosity syndrome (emergency)	Urgent plasmapheresis to rapidly lower IgM levels, followed by definitive systemic therapy

COMPLICATIONS

Disease-related:

- Hyperviscosity syndrome causing visual disturbance (retinal haemorrhage, 'sausage-link' veins), headache, confusion, and bleeding diathesis
- Peripheral neuropathy from anti-myelin-associated glycoprotein (anti-MAG) antibody activity in a subset of patients
- Bing-Neel syndrome – direct CNS infiltration by lymphoplasmacytic cells, a rare but serious complication
- Cryoglobulinaemia-related vasculitis and Raynaud phenomenon in a subset

Treatment-related:

- Rituximab-induced IgM flare acutely worsening hyperviscosity symptoms if not anticipated
- Bleeding complications from BTK inhibitor therapy compounding baseline hyperviscosity-related bleeding tendency
- Atrial fibrillation with BTK inhibitors requiring careful anticoagulation balance given bleeding risk
- Peripheral neuropathy, often disabling and only partially reversible with treatment

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Ibrutinib	First-line or relapsed symptomatic disease	420 mg orally once daily	Atrial fibrillation, bleeding risk, particularly relevant given baseline hyperviscosity-related bleeding tendency
Rituximab		375 mg/m² IV per protocol schedule	
Combination therapy; used			Transient IgM flare immediately after initiation, which can
cautiously given IgM 'flare'			worsen hyperviscosity – plasmapheresis cover considered
risk			in high-IgM patients

★ CLINICAL PEARLS & EXAM FOCUS

▸ Hyperviscosity symptoms (visual change, headache, bleeding, confusion) in a patient with known Waldenstrom's warrants urgent plasmapheresis, not simply chemotherapy alone, as a first step.

▸ Consider plasmapheresis cover before rituximab in patients with very high IgM levels to blunt the IgM flare phenomenon.

GUIDELINES & RECENT ADVANCES

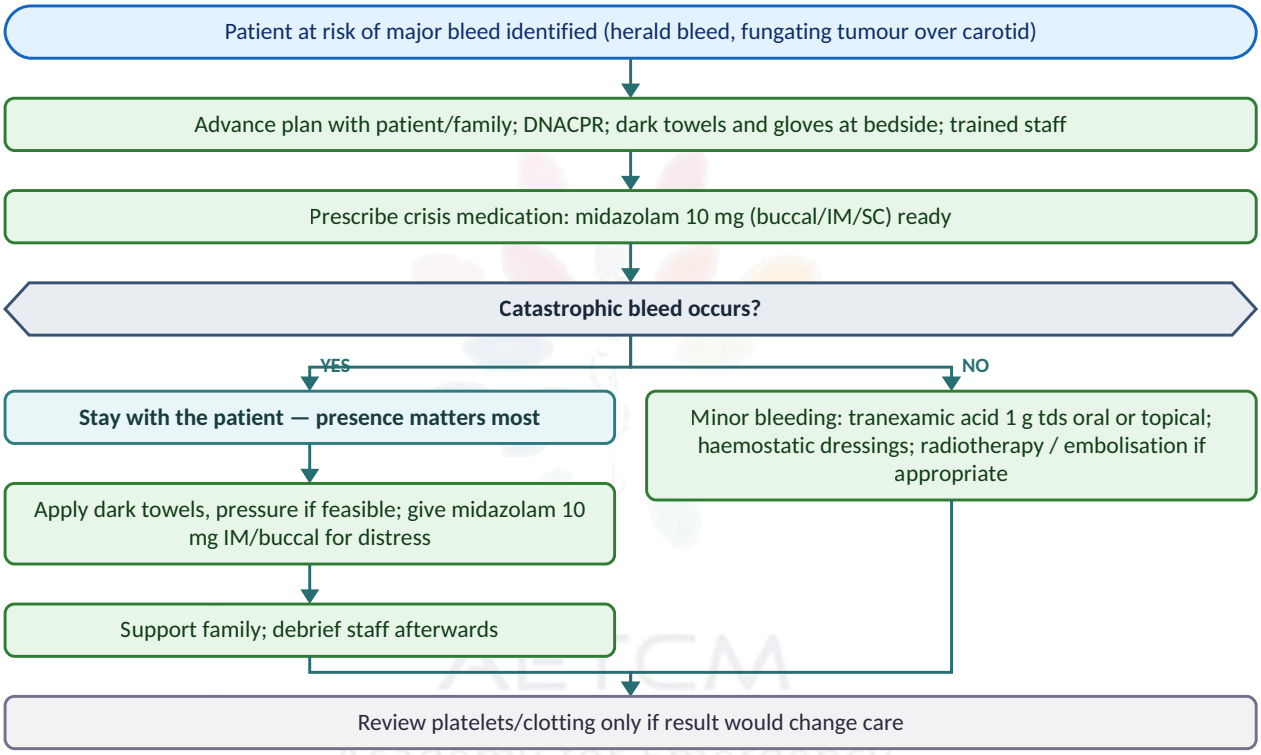
- NCCN Guidelines: Waldenstrom Macroglobulinemia/Lymphoplasmacytic Lymphoma (current version)

EXAM-ORIENTED THEORY

Palliative care focus — Waldenström macroglobulinaemia

Problem	Management
Hyperviscosity	Plasmapheresis, then rituximab-based or BTK inhibitor therapy
Peripheral neuropathy (anti-MAG)	Gabapentinoid, duloxetine
Anaemia / fatigue	Transfusion, treat disease
Cold agglutinins / cryoglobulins	Keep warm; warm IV fluids

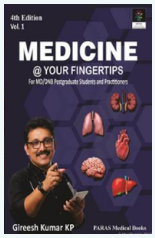
ALGORITHM · Catastrophic Haemorrhage at the End of Life Algo 20 — full page ›



★ KEY POINTS

▸ Death from catastrophic bleed is usually within minutes — sedation may not take effect; calm presence is the key intervention.

▸ Use IM route if no IV access — SC absorption is unreliable in shock.



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M63 Myelodysplastic Syndrome

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why might cytopenias temporarily worsen during early hypomethylating agent therapy? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage chronic transfusion-related complications in haematological malignancy (CD3.1, PD3)

DEFINITION A clonal haematopoietic stem cell disorder causing ineffective haematopoiesis and peripheral cytopenias despite a normo-/hypercellular marrow, with a variable risk of transformation to acute myeloid leukaemia; predominantly affects older adults.

Stage / Setting	Principal Treatment Approach
Lower-risk disease	Supportive care (transfusion, growth factors) ± lenalidomide (particularly effective in del(5q) subtype) or luspatercept for anaemia
Higher-risk disease	Hypomethylating agent therapy (azacitidine/decitabine); allogeneic stem cell transplantation in eligible, fit patients for durable disease control
Transformation to AML	Treated as secondary/therapy-related AML, generally with a worse prognosis than de novo disease

COMPLICATIONS

Disease-related:

- Chronic transfusion dependence leading to iron overload (requiring chelation therapy)
- Progressive bone marrow failure with worsening pancytopenia over time
- Transformation to acute myeloid leukaemia, the most feared complication, particularly in higher-risk disease
- Recurrent infections from neutropenia and qualitative neutrophil dysfunction

Treatment-related:

- Iron overload from chronic transfusions causing cardiac and hepatic dysfunction if chelation is not instituted
- Paradoxical worsening of cytopenias during early cycles of hypomethylating agent therapy before benefit is seen
- Graft-versus-host disease after allogeneic transplantation
- Bleeding complications from severe thrombocytopenia

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Azacitidine	Higher-risk disease	75 mg/m²/day SC/IV for 7 days, every 28 days	Myelosuppression (paradoxically can worsen cytopenias initially), injection site reactions
Lenalidomide	Lower-risk, del(5q) subtype, transfusion-dependent anaemia	10 mg orally once daily	Myelosuppression, venous thromboembolism risk, teratogenicity

★ CLINICAL PEARLS & EXAM FOCUS

► Iron chelation should be considered in chronically transfused patients, particularly those with lower-risk disease and longer expected survival, to prevent cardiac/hepatic iron overload.

► Response to hypomethylating agents may take several cycles to manifest – premature discontinuation for apparent lack of early response is a common management error.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Myelodysplastic Syndromes (current version)

M64 Polycythaemia Vera

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is the target haematocrit in polycythaemia vera and why does it matter? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply evidence-based thrombosis-prevention targets in myeloproliferative disease (CD3.1, PD3)

DEFINITION A JAK2-mutated myeloproliferative neoplasm causing clonal overproduction of red cells (and often white cells/platelets), presenting with plethora, pruritus (classically aquagenic, after warm water exposure), headache, or a thrombotic event.

Stage / Setting	Principal Treatment Approach
Low-risk (age <60, no prior thrombosis)	Low-dose aspirin and therapeutic phlebotomy to maintain haematocrit below the target threshold
High-risk (age ≥60 or prior thrombosis)	Cytoreductive therapy (hydroxyurea first-line) added to phlebotomy and aspirin
Hydroxyurea-resistant/intolerant disease	JAK inhibitor (ruxolitinib) as an alternative cytoreductive agent

COMPLICATIONS

Disease-related:

- Arterial and venous thrombosis (stroke, myocardial infarction, Budd-Chiari syndrome, splanchnic vein thrombosis) – the dominant cause of morbidity/mortality
- Severe, often debilitating aquagenic pruritus
- Progression to myelofibrosis ('post-polycythaemic myelofibrosis') over years
- Progression to acute myeloid leukaemia, an uncommon but recognised late event

Treatment-related:

- Herpes zoster reactivation with ruxolitinib therapy
- Hydroxyurea-related leg ulcers, sometimes chronic and treatment-limiting
- Rebound symptoms (fever, severe splenomegaly flare) if ruxolitinib is stopped abruptly, requiring gradual tapering
- Bleeding paradoxically occurring alongside thrombotic risk due to acquired platelet dysfunction at very high platelet counts

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Hydroxyurea	First-line cytoreductive therapy, high-risk disease	500-1000 mg orally daily, titrated to blood counts	Myelosuppression, mucocutaneous ulcers, rare leg ulcers, long-term leukaemogenic potential debated
Ruxolitinib			

Trauma & Critical care Medicine

Hydroxyurea resistance/ intolerance	10 mg orally twice daily, titrated	Cytopenias, infection risk (including herpes zoster reactivation), withdrawal rebound symptoms if stopped abruptly

★ CLINICAL PEARLS & EXAM FOCUS

▸ Maintaining haematocrit below target (generally <45%) through phlebotomy is the single most evidence-based intervention for reducing thrombotic risk.

▸ Aquagenic pruritus (worsening after bathing) is a classic, frequently examined diagnostic clue for polycythaemia vera.

GUIDELINES & RECENT ADVANCES

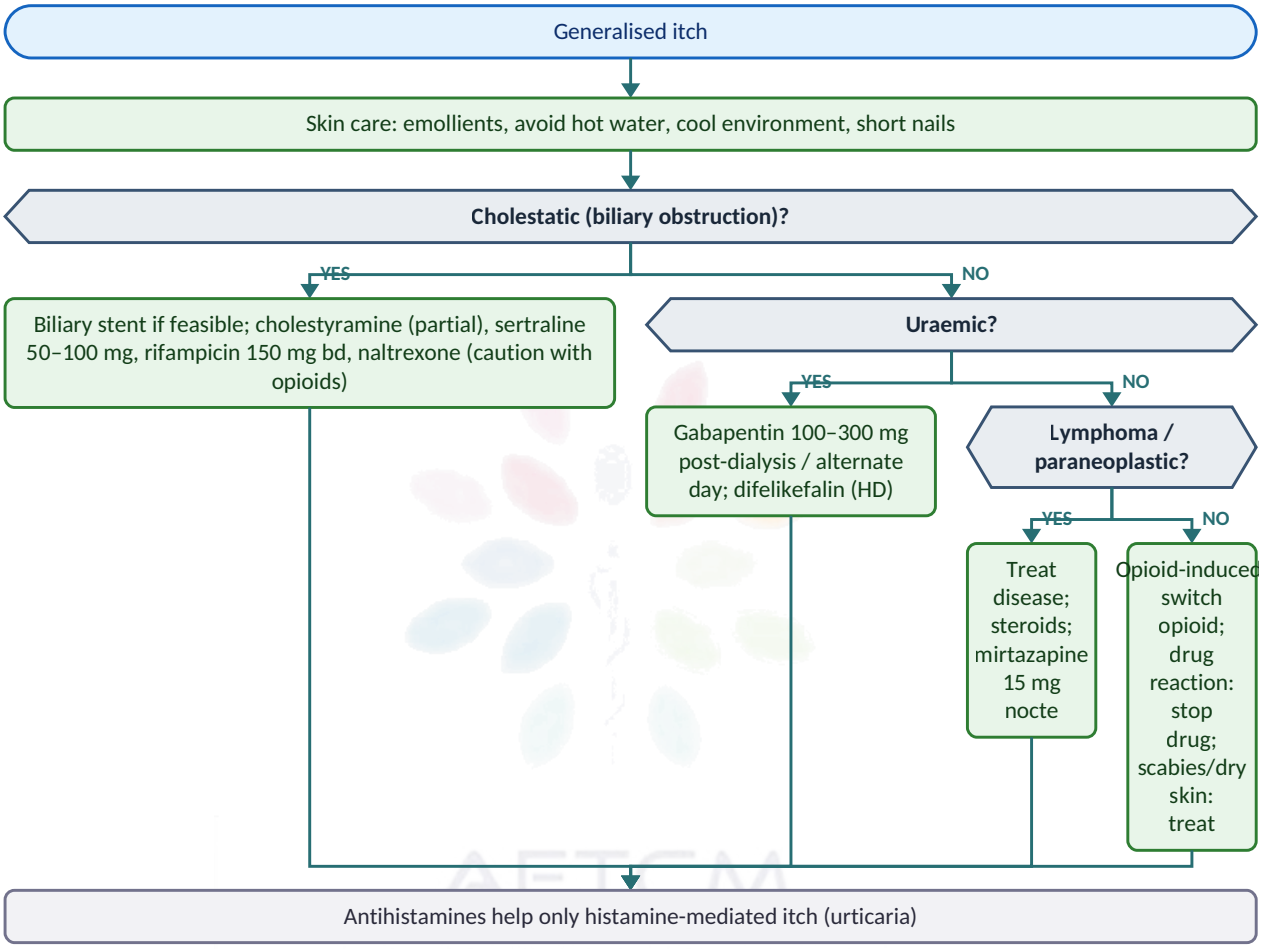
- NCCN Guidelines: Myeloproliferative Neoplasms (current version)

EXAM-ORIENTED THEORY

Palliative care focus — polycythaemia vera

Problem	Management
Aquagenic pruritus	SSRIs (paroxetine), antihistamines, ruxolitinib
Thrombosis risk	Venesection to Hct < 45%, low-dose aspirin, hydroxyurea
Erythromelalgia	Aspirin
Progression to myelofibrosis / AML	Splenomegaly pain, cachexia — ruxolitinib, symptom care

ALGORITHM · Pruritus in Palliative Care Algo 57 — full page ›



★ KEY POINTS

- ▸ Opioid antagonists can precipitate pain and withdrawal in patients on opioids.
- ▸ Sertraline and mirtazapine are useful in mixed causes.

M65 Essential Thrombocythaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why can very high platelet counts paradoxically increase bleeding risk? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply mutation-based risk stratification in myeloproliferative neoplasms (CD2.1, CD4.1)

DEFINITION A myeloproliferative neoplasm causing clonal overproduction of platelets, driven by JAK2, CALR or MPL mutations in most cases; often asymptomatic and discovered incidentally, but carries thrombotic and hemorrhagic risk disproportionate to the platelet count alone.

Stage / Setting	Principal Treatment Approach
Low-risk (age <60, no prior thrombosis, no high-risk mutation)	Low-dose aspirin alone, with observation; cytoreduction generally not needed
High-risk (age ≥60, prior thrombosis, or JAK2-mutated with additional risk factors)	Cytoreductive therapy (hydroxyurea first-line) added to aspirin
Hydroxyurea-resistant/intolerant or pregnancy	Interferon-alfa (preferred in pregnancy and younger patients) or anagrelide as alternatives

COMPLICATIONS

Disease-related:

- Thrombotic events (arterial more than venous), including stroke, myocardial infarction and digital ischaemia (erythromelalgia)
- Paradoxical bleeding at very high platelet counts due to acquired von Willebrand syndrome
- Progression to myelofibrosis or, less commonly, acute leukaemia over long-term follow-up
- Pregnancy complications (miscarriage, placental insufficiency) in affected women of childbearing age

Treatment-related:

- Hydroxyurea-related mucocutaneous ulcers and, rarely, leg ulcers
- Cardiac side effects (palpitations, arrhythmia) with anagrelide, requiring caution in older patients or those with cardiac disease
- Teratogenicity of hydroxyurea, necessitating interferon-alfa as the preferred cytoreductive agent in pregnancy
- Bleeding risk from acquired von Willebrand syndrome at extreme thrombocytosis, sometimes underappreciated

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Hydroxyurea	First-line cytoreduction, high-risk disease	500-1500 mg orally daily, titrated to platelet count	Myelosuppression, mucocutaneous ulcers
Anagrelide	Second-line cytoreduction	0.5 mg orally twice daily, titrated	Palpitations, fluid retention, headache – caution in cardiac disease

★ CLINICAL PEARLS & EXAM FOCUS

► Platelet count alone does not predict risk – mutation status (JAK2 vs CALR vs triple-negative) and age/thrombosis history drive risk stratification and treatment decisions.

► At very high platelet counts, paradoxical bleeding risk (acquired von Willebrand syndrome) should be considered, particularly before surgery or invasive procedures.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Myeloproliferative Neoplasms (current version)

M66 Primary Myelofibrosis

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must ruxolitinib be tapered rather than abruptly discontinued? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage symptom burden and withdrawal phenomena in myelofibrosis (CD3.1, PD3)

DEFINITION The most clinically severe of the classic myeloproliferative neoplasms, characterised by progressive bone marrow fibrosis, extramedullary haematopoiesis (massive splenomegaly), and constitutional symptoms; may arise de novo or follow polycythaemia vera/essential thrombocythaemia.

Stage / Setting	Principal Treatment Approach
Low-risk, asymptomatic	Observation alone, given generally indolent course in this risk group
Symptomatic, intermediate/high-risk, transplant-ineligible	JAK inhibitor (ruxolitinib) for splenomegaly and constitutional symptom control
Transplant-eligible, high-risk disease	Allogeneic stem cell transplantation, the only potentially curative option

COMPLICATIONS

Disease-related:

- Massive, often painful splenomegaly causing early satiety, abdominal discomfort and cytopenias from hypersplenism
- Severe constitutional symptoms (fatigue, night sweats, weight loss, bone pain) significantly impairing quality of life
- Progressive marrow failure with transfusion-dependent anaemia
- Transformation to acute myeloid leukaemia, a major cause of mortality

Treatment-related:

- Rebound symptom flare (fever, rapid splenomegaly recurrence) if ruxolitinib is stopped abruptly – must be tapered, not stopped suddenly
- Herpes zoster and other infections with JAK inhibitor-related immunosuppression
- Graft-versus-host disease after allogeneic transplantation, the only curative but high-risk option
- Splenic infarction, causing severe left upper quadrant pain, in markedly enlarged spleens

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Ruxolitinib	Symptomatic splenomegaly/ constitutional symptoms	5-20 mg orally twice daily, titrated to platelet count/ response	Cytopenias, infection risk (herpes zoster, atypical infections), rebound symptoms on abrupt discontinuation
Danazol	Anaemia management, adjunct therapy	200 mg orally 2-3 times daily	Hepatotoxicity, virilisation, fluid retention

★ CLINICAL PEARLS & EXAM FOCUS

▸ Symptom burden (fatigue, night sweats, splenomegaly-related discomfort) can be profound even when blood counts look relatively stable – symptom-directed palliative input is valuable throughout the disease course, not only at end of life.

▸ Ruxolitinib must be tapered rather than stopped abruptly, given a well-recognised, sometimes severe withdrawal/rebound symptom flare.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Myeloproliferative Neoplasms (current version)

M67 Chronic Myelomonocytic Leukaemia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How does treatment selection in CMML depend on its myelodysplastic vs myeloproliferative features? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Classify overlap haematological syndromes to guide individualised therapy (CD2.1, CD4.1)

DEFINITION A clonal disorder with overlapping myelodysplastic and myeloproliferative features, defined by persistent peripheral blood monocytosis, occurring predominantly in older adults and carrying a variable but often significant risk of transformation to acute myeloid leukaemia.

Stage / Setting	Principal Treatment Approach
Myelodysplastic-predominant, lower-risk	Supportive care (transfusion support) ± hypomethylating agent therapy for symptomatic cytopenias
Myeloproliferative-predominant (leucocytosis-driven)	Cytoreductive therapy (hydroxyurea) to control white cell count and organomegaly
Higher-risk disease, transplant-eligible	Allogeneic stem cell transplantation, the only potentially curative approach

COMPLICATIONS

Disease-related:

- Progressive cytopenias with recurrent infection and bleeding risk
- Organomegaly (hepatosplenomegaly) from monocytic infiltration
- Transformation to acute myeloid leukaemia, a major and relatively common complication
- Extramedullary infiltration (skin, lymph nodes) by monocytic cells in a subset of patients

Treatment-related:

- Prolonged myelosuppression from both hydroxyurea and hypomethylating agent therapy
- Graft-versus-host disease after allogeneic transplantation
- Treatment-related cytopenias complicating already compromised baseline marrow function
- Iron overload from chronic transfusion dependence over the disease course

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Hydroxyurea	Myeloproliferative-predominant disease with leucocytosis	500-2000 mg orally daily, titrated to blood counts	Myelosuppression, mucocutaneous ulceration
Azacitidine	Myelodysplastic-predominant or higher-risk disease	75 mg/m²/day SC for 7 days, every 28 days	Myelosuppression, injection site reactions

★ CLINICAL PEARLS & EXAM FOCUS

▸ CMML sits at the overlap of myelodysplastic and myeloproliferative classification – treatment choice depends on which phenotype (cytopenic vs proliferative) predominates clinically.

▸ Given the heterogeneous and often prolonged disease course, integrating supportive/palliative care alongside disease-directed therapy from diagnosis is appropriate.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Myelodysplastic/Myeloproliferative Neoplasms (current version)

M68 Cutaneous Malignant Melanoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Compare the relative advantages of immunotherapy versus targeted therapy in BRAF-mutant metastatic melanoma. [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply biomarker-driven sequencing decisions in advanced melanoma (CD2.1, CD4.1)

DEFINITION The most lethal common skin cancer, arising from melanocytes, with incidence rising in fair-skinned populations linked to UV exposure; early detection via the ABCDE criteria (Asymmetry, Border, Colour, Diameter, Evolution) dramatically improves outcomes, as depth of invasion (Breslow thickness) is the key prognostic factor.

Stage / Setting	Principal Treatment Approach
Early, thin (localised)	Wide local excision with margin based on Breslow thickness; sentinel lymph node biopsy for intermediate/ thick lesions
Locally advanced/nodal	Wide excision with completion lymphadenectomy or nodal observation, plus adjuvant immunotherapy (anti-PD-1) or targeted therapy (if BRAF-mutated) for high-risk resected disease
Metastatic	BRAF/MEK inhibitor combination if BRAF V600-mutated, or checkpoint inhibitor immunotherapy (nivolumab-ipilimumab or anti-PD-1 monotherapy) otherwise

COMPLICATIONS

Disease-related:

- Rapid lymphatic and haematogenous spread to brain, liver, lung and bone, often simultaneously involving multiple organs
- Brain metastases, disproportionately common and sometimes haemorrhagic
- In-transit metastases (satellite skin/subcutaneous nodules between primary site and regional nodes)
- Ulceration of the primary lesion, an adverse prognostic feature

Treatment-related:

- Severe immune-related colitis/hepatitis with combination checkpoint inhibitor therapy, sometimes requiring high-dose steroids or biologic immunosuppression
- Pyrexia syndrome with dabrafenib, occasionally necessitating treatment interruption
- Lymphoedema after completion lymphadenectomy
- Secondary cutaneous squamous cell carcinomas, paradoxically, with some BRAF inhibitor monotherapy regimens (reduced with combination MEK inhibition)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Nivolumab + Ipilimumab	First-line metastatic disease, across BRAF status	Nivolumab 1 mg/kg + ipilimumab 3 mg/kg every 3	Severe immune-related adverse events, notably colitis and hepatitis,
		weeks x4, then nivolumab maintenance	more frequent with combination than monotherapy
Dabrafenib + Trametinib	BRAF V600E/K-mutated disease	Dabrafenib 150 mg twice daily + trametinib 2 mg once daily	Fever (dabrafenib), rash, cardiomyopathy (trametinib) – rapid but sometimes less durable response than immunotherapy

★ CLINICAL PEARLS & EXAM FOCUS

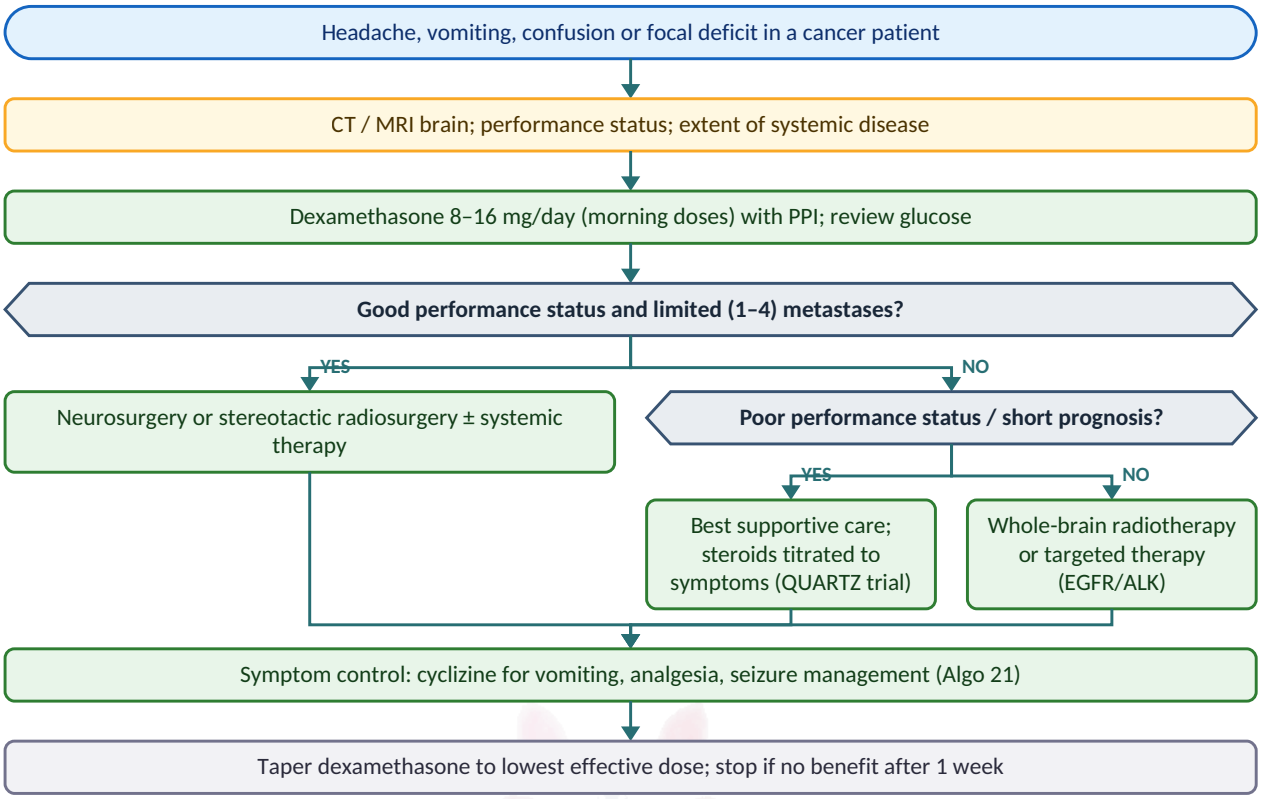
▸ Immunotherapy produces more durable long-term responses than targeted therapy in BRAF-mutant disease, though targeted therapy acts faster – sequencing strategy is an evolving, frequently examined area.

▸ The ABCDE criteria remain the key public-health teaching tool for early melanoma detection.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Cutaneous Melanoma (current version)

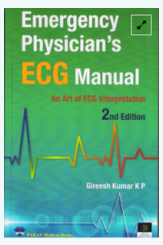
ALGORITHM · Raised Intracranial Pressure and Brain Metastases Algo 22 — full page ›



★ KEY POINTS

- ▸ QUARTZ: WBRT added little to quality-adjusted survival in poor-prognosis NSCLC brain metastases.
- ▸ Give steroids before 2 pm to reduce insomnia.

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M69 Basal Cell Carcinoma

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is Mohs micrographic surgery preferred for facial basal cell carcinoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Select tissue-conserving surgical techniques for cosmetically sensitive malignancy sites (PD3, PD8)

DEFINITION The commonest skin cancer overall, arising from basal keratinocytes, locally invasive but with very low metastatic potential; typically presents as a pearly, telangiectatic papule or non-healing ulcer on sun-exposed skin, especially the face.

Stage / Setting	Principal Treatment Approach
Low-risk, localised	Surgical excision with clear margins, or Mohs micrographic surgery for cosmetically/ functionally sensitive sites
High-risk or recurrent	Mohs micrographic surgery preferred for maximal tissue conservation with margin control
Locally advanced/ metastatic (rare)	Hedgehog pathway inhibitor (vismodegib/sonidegib) or immunotherapy (cemiplimab) for unresectable/metastatic disease

COMPLICATIONS

Disease-related:

- Local tissue destruction causing disfigurement, particularly on the face (eyes, nose, ears)
- Rarely, deep invasion into bone/cartilage ('rodent ulcer' behaviour) if neglected
- Nerve invasion causing pain or sensory/motor deficit in advanced local disease
- Metastasis is rare but can occur in long-neglected, extensive disease

Treatment-related:

- Cosmetic/functional disfigurement after excision of facial lesions, sometimes requiring complex reconstruction
- Hedgehog inhibitor-related muscle cramps and taste disturbance, often treatment-limiting due to quality-of-life impact
- Local recurrence, particularly with infiltrative/morphoeic histological subtypes
- Multiple primary basal cell carcinomas over a lifetime in chronically sun-exposed/fair-skinned individuals

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Vismodegib	Locally advanced/metastatic, unresectable disease	150 mg orally once daily	Muscle spasms, dysgeusia, alopecia, teratogenicity (contraception mandatory)

★ CLINICAL PEARLS & EXAM FOCUS

▸ Despite being 'low-grade', neglected basal cell carcinoma can cause major local tissue destruction – early treatment prevents disfiguring outcomes.

▸ Mohs micrographic surgery offers the highest cure rate with maximal normal tissue preservation for facial lesions.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Basal Cell Skin Cancer (current version)

M70 Cutaneous Squamous Cell Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is caution needed before using immunotherapy in a solid organ transplant recipient? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise immunotherapy contraindications relevant to transplant history (CD4.1, PD2)

DEFINITION The second commonest skin cancer, arising from keratinocytes, with higher metastatic potential than basal cell carcinoma, particularly in immunosuppressed patients (transplant recipients), on the lip/ear, or arising in chronic wounds/scars (Marjolin's ulcer).

Stage / Setting	Principal Treatment Approach
Low-risk, localised	Surgical excision with adequate margins
High-risk features (perineural invasion, immunosuppression, recurrent, high-risk site)	Mohs micrographic surgery or wide excision with adjuvant radiotherapy
Locally advanced/metastatic	Immunotherapy (cemiplimab/pembrolizumab) as the preferred systemic option; platinum-based chemotherapy as an alternative

COMPLICATIONS

Disease-related:

- Perineural invasion causing pain and risk of skull-base spread in head/neck lesions
- Regional lymph node metastasis, particularly from lip, ear, or immunosuppression-related tumours
- Marjolin's ulcer – aggressive SCC arising in a chronic burn scar or non-healing wound
- Local tissue destruction with bone/cartilage invasion in neglected disease

Treatment-related:

- Graft rejection risk if checkpoint inhibitor immunotherapy is used in a solid organ transplant recipient – a critical, frequently examined caution
- Surgical defect reconstruction challenges in high-risk anatomical sites (ear, lip, periorbital)
- Local recurrence with inadequately excised or high-risk histological subtypes
- Nerve-related pain/dysfunction from perineural spread

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cemiplimab	Locally advanced/metastatic, unresectable disease	350 mg IV every 3 weeks	Immune-related adverse events; caution in solid organ transplant recipients (rejection risk)

★ CLINICAL PEARLS & EXAM FOCUS

▸ Immunotherapy carries a real risk of precipitating organ transplant rejection – transplant history must always be elicited before considering checkpoint inhibitors.

▸ Any chronic non-healing wound or burn scar developing a new nodule or ulceration should raise suspicion for Marjolin's ulcer (SCC).

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Squamous Cell Skin Cancer (current version)

M71 Merkel Cell Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is sentinel lymph node biopsy recommended even for small Merkel cell carcinomas? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise disproportionate metastatic risk relative to primary tumour size (CD4.1)

DEFINITION A rare, highly aggressive neuroendocrine skin cancer associated with Merkel cell polyomavirus infection and chronic UV exposure/immunosuppression, typically presenting as a rapidly growing, painless, red-violet nodule, often on sun-exposed skin of older adults.

Stage / Setting	Principal Treatment Approach
Localised	Wide local excision with sentinel lymph node biopsy, given high rates of occult nodal metastasis even in small primaries
Nodal disease	Lymphadenectomy and/or radiotherapy to the nodal basin, given marked radiosensitivity of this tumour
Metastatic/ unresectable	Immunotherapy (avelumab/pembrolizumab) as first-line, given notably high response rates

COMPLICATIONS

Disease-related:

- High rate of occult regional lymph node metastasis even from small primary lesions
- Rapid local recurrence if margins are inadequate
- Distant metastasis (liver, bone, brain, lung) in advanced disease
- Immunosuppression (HIV, transplant, CLL) as both a risk factor and a complicating factor for aggressive behaviour

Treatment-related:

- Radiation-induced skin changes given the marked radiosensitivity-driven reliance on radiotherapy
- Immune-related toxicity with checkpoint inhibitors, particularly relevant given frequent baseline immunosuppression in this population
- Wound complications after wide excision and sentinel node biopsy in often elderly, comorbid patients
- Rapid recurrence requiring vigilant short-interval follow-up

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Avelumab	Metastatic disease, first-line	800 mg IV every 2 weeks	Immune-related adverse events, infusion reactions

★ CLINICAL PEARLS & EXAM FOCUS

► Despite small size, Merkel cell carcinoma carries a disproportionately high risk of occult nodal spread – sentinel node biopsy should not be omitted based on primary tumour size alone.

► Immunotherapy response rates are notably high in this cancer, making it a good example of immune-responsive virally-associated malignancy.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Merkel Cell Carcinoma (current version)

M72 Kaposi Sarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why might Kaposi sarcoma lesions worsen shortly after starting antiretroviral therapy? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise immune reconstitution inflammatory syndrome in HIV-associated malignancy (CD4.1, CD9)

DEFINITION A vascular tumour caused by human herpesvirus-8 (HHV-8), occurring in classic (elderly Mediterranean/ Jewish men, indolent), endemic (African), iatrogenic (transplant-related immunosuppression) and epidemic (HIV/AIDS-associated, often aggressive) forms; presents as violaceous skin/mucosal nodules or plaques.

Stage / Setting	Principal Treatment Approach
Classic, limited skin disease	Local therapy – radiotherapy, topical/intralesional treatment, or observation given indolent course
Epidemic (HIV-associated)	Antiretroviral therapy is the cornerstone, often causing regression; systemic chemotherapy added for extensive/visceral disease
Iatrogenic (transplant-related)	Reduction of immunosuppression as the first step, ± conversion to an mTOR inhibitor-based immunosuppressive regimen (which has independent antitumour activity)

COMPLICATIONS

Disease-related:

- Mucosal and GI tract involvement causing bleeding or obstruction
- Pulmonary Kaposi sarcoma causing breathlessness and haemoptysis, a poor prognostic feature
- Lymphoedema from lymphatic obstruction by tumour, particularly of the lower limbs
- Disfiguring, stigmatising skin lesions with significant psychosocial impact, especially in HIV-associated disease

Treatment-related:

- Immune reconstitution inflammatory syndrome (IRIS) causing paradoxical worsening of Kaposi sarcoma lesions shortly after starting antiretroviral therapy
- Cardiotoxicity, though reduced, still a consideration with liposomal doxorubicin
- Stigma and psychosocial distress, particularly significant given the HIV association in epidemic disease
- Lymphoedema-related skin breakdown and secondary infection

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Liposomal Doxorubicin	Extensive/visceral/ symptomatic disease	20 mg/m² IV every 2-3 weeks	Cardiotoxicity (reduced relative to conventional doxorubicin), palmar-plantar erythrodysesthesia, myelosuppression
Antiretroviral Therapy	Foundation of treatment in HIV- associated disease	Per standard HIV treatment guidelines	Immune reconstitution inflammatory syndrome can transiently worsen Kaposi sarcoma lesions

★ CLINICAL PEARLS & EXAM FOCUS

► Antiretroviral therapy alone can induce regression of HIV-associated Kaposi sarcoma – always optimise HIV control before/alongside considering additional chemotherapy.

► IRIS-related flare after starting antiretrovirals should be anticipated and explained to patients, not mistaken for treatment failure.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Kaposi Sarcoma / AIDS-related malignancies resources

M73 Angiosarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is Stewart-Treves syndrome? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise late complications of chronic lymphoedema and prior radiotherapy (CD4.1, CD9)

DEFINITION A rare, aggressive malignancy of vascular/lymphatic endothelial origin, occurring in the skin of the elderly scalp/face, in a chronically lymphoedematous limb (Stewart-Treves syndrome, classically post-mastectomy), or as a radiation-induced secondary malignancy in previously irradiated breast/soft tissue.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Wide surgical excision with clear margins, often challenging given infiltrative, multifocal growth pattern
Locally advanced/ unresectable	Radiotherapy ± systemic chemotherapy (taxane-based) for disease control
Metastatic	Taxane-based or anthracycline-based chemotherapy; antiangiogenic agents (pazopanib) given the vascular tumour biology

COMPLICATIONS

Disease-related:

- Multifocal skin/soft tissue involvement making complete surgical clearance difficult
- Spontaneous bleeding from friable, highly vascular tumour nodules
- Rapid local recurrence despite apparently clear margins, given a field-change/multifocal growth pattern
- Distant metastasis (lung, liver) in advanced disease

Treatment-related:

- Bleeding complications, both spontaneous from the tumour and treatment-related with antiangiogenic therapy
- Wound healing difficulty after excision of extensively infiltrated or previously irradiated skin
- Radiotherapy limitations in previously irradiated fields (radiation-induced angiosarcoma)
- Psychosocial distress from disfiguring, often bleeding skin lesions

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Paclitaxel	First-line metastatic/ unresectable disease	80 mg/m² weekly (3 weeks on/1 week off) or 175 mg/m² every 3 weeks	Peripheral neuropathy, myelosuppression
Pazopanib	Relapsed/refractory disease, given vascular tumour biology	800 mg orally once daily	Hypertension, hepatotoxicity, bleeding risk given anti-angiogenic mechanism

★ CLINICAL PEARLS & EXAM FOCUS

▸ Chronic lymphoedema of any cause carries a long-term risk of angiosarcoma (Stewart-Treves syndrome) – a new nodule or bruise-like lesion in a chronically lymphoedematous limb warrants biopsy.

▸ Radiation-induced angiosarcoma of the breast, arising years after radiotherapy for breast cancer, is an important late complication to recognise.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Soft Tissue Sarcoma (current version)

M74 Glioblastoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role and complications of corticosteroid therapy in glioblastoma management. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Balance symptom control with steroid-related toxicity in neuro-oncology (CD3.1, PD3)

DEFINITION The commonest and most aggressive primary malignant brain tumour in adults (WHO grade 4 astrocytoma), characterised by rapid growth, extensive infiltration beyond visible margins, and a poor prognosis despite maximal therapy; presents with headache, seizures, or progressive focal neurological deficit.

Stage / Setting	Principal Treatment Approach
Newly diagnosed, good performance status	Maximal safe surgical resection followed by concurrent chemoradiation (temozolomide-radiotherapy) and adjuvant temozolomide
Newly diagnosed, poor performance status/elderly	Hypofractionated radiotherapy ± temozolomide, or best supportive care depending on overall status
Recurrent disease	Re-resection where feasible, bevacizumab for symptom palliation (reduces oedema/ steroid requirement), or re-irradiation; tumour-treating fields in selected cases

COMPLICATIONS

Disease-related:

- Raised intracranial pressure causing headache, vomiting and declining consciousness
- Seizures, common both at presentation and through the disease course
- Progressive focal neurological deficit (hemiparesis, aphasia, visual field loss) from tumour growth/ infiltration
- Rapid, near-universal recurrence given diffusely infiltrative growth beyond resection margins

Treatment-related:

- Steroid-related complications (hyperglycaemia, myopathy, osteoporosis, immunosuppression/ opportunistic infection) from prolonged dexamethasone use
- Pneumocystis jirovecii pneumonia risk during concurrent temozolomide-radiotherapy given treatment-induced lymphopenia
- Cognitive decline from both tumour progression and whole-brain radiation effects
- Severe psychosocial and caregiver burden given rapid functional decline

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Temozolomide	Concurrent and adjuvant chemotherapy	75 mg/m²/day with radiotherapy, then 150-200 mg/ m²/day x5 days every 28 days (adjuvant)	Myelosuppression, nausea – requires Pneumocystis prophylaxis during concurrent phase given lymphopenia risk
Dexamethasone	Peritumoural oedema control, throughout disease course	4-16 mg/day in divided doses, tapered to lowest effective dose	Hyperglycaemia, proximal myopathy, immunosuppression, mood disturbance with prolonged use
Bevacizumab	Recurrent disease, symptom/oedema palliation	10 mg/kg IV every 2 weeks	Hypertension, bleeding, impaired wound healing, bowel perforation (rare)

★ CLINICAL PEARLS & EXAM FOCUS

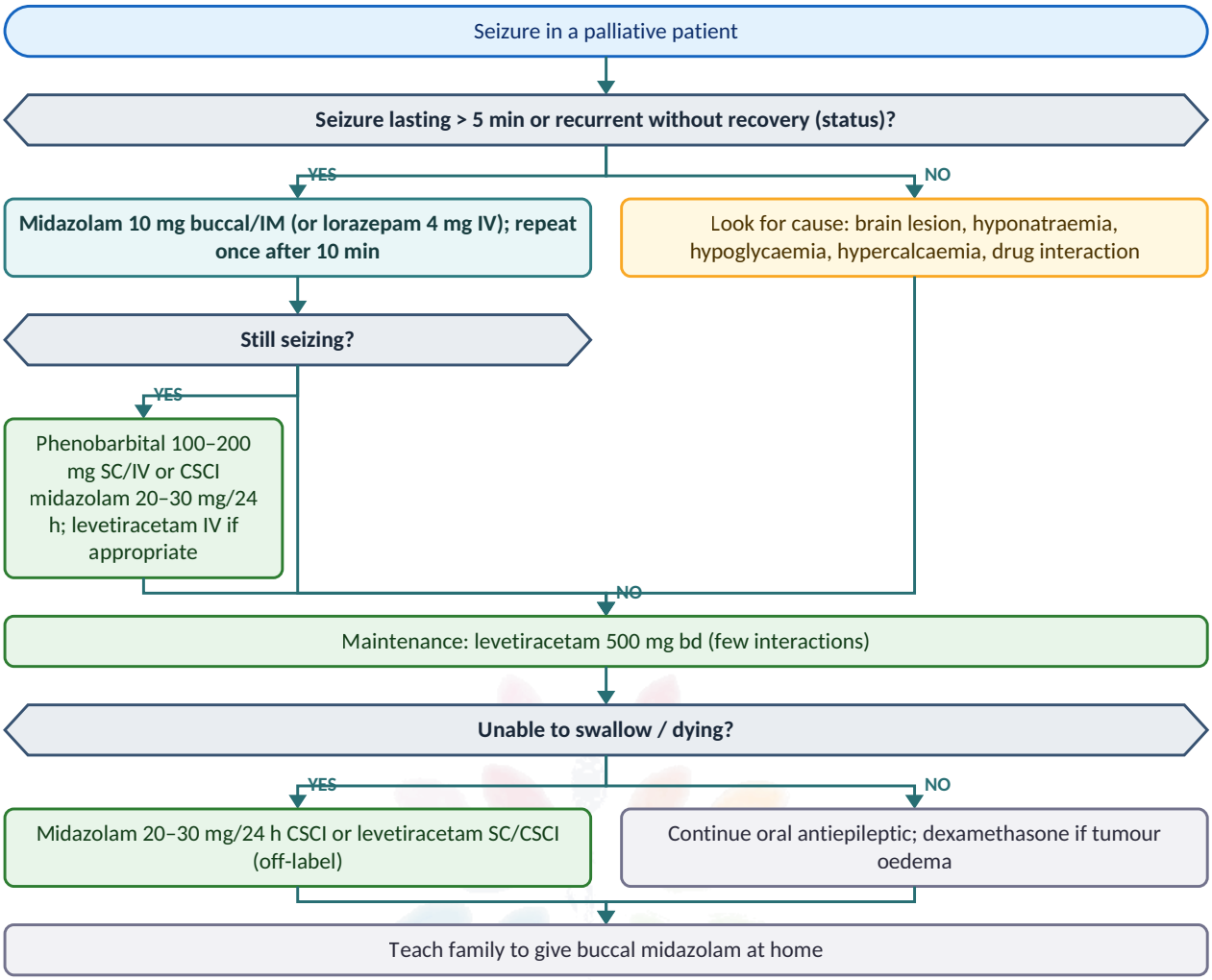
▸ Steroids should be tapered to the lowest effective dose as soon as clinically feasible, given their substantial cumulative toxicity burden over a disease course measured in months.

▸ Early, proactive palliative care integration is strongly recommended given glioblastoma's near-uniformly poor prognosis and heavy symptom/caregiver burden.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Central Nervous System Cancers (current version)

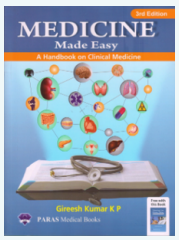
ALGORITHM · Seizures in Palliative Care Algo 21 — full page ›



★ KEY POINTS

- ▸ Avoid enzyme-inducing antiepileptics (phenytoin, carbamazepine) with chemotherapy and dexamethasone.
- ▸ No primary seizure prophylaxis for brain metastases without seizures.

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M75 Diffuse (Low-Grade) Astrocytoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is IDH mutation testing important in diffuse astrocytoma management? [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply molecular classification to prognosis counselling in low-grade glioma (CD2.1, AD2)

DEFINITION A WHO grade 2 glial tumour, typically occurring in younger adults, presenting most often with seizures given its frequent cortical/temporal location; although lower-grade than glioblastoma, it is not benign and eventually transforms to a higher-grade tumour in most cases over years.

Stage / Setting	Principal Treatment Approach
Small, surgically accessible	Maximal safe resection; molecular profiling (IDH mutation, 1p/19q co-deletion) to refine prognosis and further management
Residual/high-risk features after surgery	Adjuvant radiotherapy combined with PCV or temozolomide chemotherapy
Malignant transformation to higher grade	Managed as the corresponding higher-grade glioma (treated as glioblastoma if transformed to grade 4)

COMPLICATIONS

Disease-related:

- Seizures, often the presenting and persisting symptom even after treatment
- Progressive cognitive and personality change from tumour growth/infiltration, particularly with frontal/temporal location
- Malignant transformation to a higher-grade glioma, usually the eventual cause of death
- Mass effect and raised intracranial pressure as the tumour enlarges over time

Treatment-related:

- Antiepileptic drug-related side effects (mood/behavioural change, cognitive slowing) requiring careful medication selection and monitoring
- Radiation-induced cognitive decline, a significant long-term concern given younger patient age and longer expected survival than higher-grade gliomas
- Post-surgical neurological deficit depending on tumour location and extent of resection
- Psychosocial impact of living for years with a known 'low-grade but not benign' brain tumour diagnosis

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Temozolomide	Adjuvant therapy for high-risk low-grade glioma	150-200 mg/m²/day x5 days every 28 days	Myelosuppression, nausea
Levetiracetam	Seizure control, throughout disease course	500-1500 mg orally twice daily, titrated	Mood/behavioural disturbance, somnolence

★ CLINICAL PEARLS & EXAM FOCUS

▸ IDH mutation status is strongly prognostic (favourable) and increasingly targetable – molecular testing should be routine, not optional, in diffuse glioma.

▸ Patients should be counselled that 'low-grade' does not mean benign – most tumours eventually progress, which shapes long-term surveillance and planning.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Central Nervous System Cancers (current version)

M76 Oligodendroglioma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What molecular marker predicts chemosensitivity in oligodendroglioma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Link co-deletion status to treatment response and prognosis (CD2.1, CD4.1)

DEFINITION A glial tumour defined by IDH mutation combined with 1p/19q co-deletion, generally more chemosensitive and better-prognosis than astrocytic tumours of similar grade, often presenting with seizures in young to middle-aged adults.

Stage / Setting	Principal Treatment Approach
Grade 2, favourable features	Maximal safe resection; observation or adjuvant therapy deferred in very favourable, completely resected cases
Grade 2 with high-risk features, or Grade 3	Surgery followed by radiotherapy with PCV (procarbazine-lomustine-vincristine) or temozolomide chemotherapy
Recurrent disease	Further chemotherapy (given characteristic chemosensitivity) or re-irradiation depending on prior treatment

COMPLICATIONS

Disease-related:

- Seizures, frequently the presenting and a persisting symptom throughout the disease course
- Progressive neurological deficit as the tumour enlarges or transforms to higher grade
- Malignant transformation to a higher-grade (anaplastic) tumour over time
- Haemorrhage into the tumour, an uncommon but recognised complication given vascularity

Treatment-related:

- Myelosuppression from PCV chemotherapy, sometimes cumulative and dose-limiting
- Peripheral neuropathy from vincristine, cumulative with repeated cycles
- Antiepileptic-related side effects requiring long-term medication management
- Cognitive effects of radiotherapy, relevant given generally favourable long-term survival in this tumour type

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Procarbazine + Lomustine + Vincristine (PCV)	Adjuvant chemotherapy, particularly effective in 1p/19q co-deleted tumours	Per standard 6-8 week cycle protocol	Myelosuppression (lomustine/ procarbazine), peripheral neuropathy (vincristine), pulmonary toxicity (rare, procarbazine)
Temozolomide	Alternative, better- tolerated adjuvant chemotherapy option	150-200 mg/ m^2/ day x5 days every 28 days	Myelosuppression, nausea

★ CLINICAL PEARLS & EXAM FOCUS

▸ 1p/19q co-deletion predicts strong chemosensitivity, particularly to PCV – a key molecular-prognostic teaching point distinguishing oligodendroglioma from astrocytoma.

▸ Given relatively long survival, minimising cumulative long-term treatment toxicity (cognitive, neuropathic) is a central management consideration.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Central Nervous System Cancers (current version)

M77 Meningioma (Atypical/Malignant)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How does meningioma grade influence recurrence risk and management? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply grade-based surveillance and treatment planning in meningioma (CD4.1, AD3)

DEFINITION Meningiomas arise from the meninges and are usually benign (WHO grade 1), but atypical (grade 2) and malignant/anaplastic (grade 3) variants exist with higher recurrence rates and, rarely, distant metastatic potential; presentation depends heavily on location (seizures, focal deficit, raised intracranial pressure).

Stage / Setting	Principal Treatment Approach
Grade 1, asymptomatic, small	Observation with serial imaging, as many never require intervention
Grade 1, symptomatic/growing, or any Grade 2-3	Maximal safe surgical resection
Grade 2-3 (atypical/malignant), or incompletely resected	Adjuvant radiotherapy given higher recurrence risk compared with grade 1 disease

COMPLICATIONS

Disease-related:

- Seizures from cortical irritation, particularly with convexity/parasagittal tumours
- Focal neurological deficit from mass effect on adjacent brain or cranial nerves (e.g. visual loss with optic nerve sheath/sphenoid wing meningioma)
- Venous sinus invasion/occlusion causing raised intracranial pressure
- Rare distant metastasis (lung, bone) in malignant/anaplastic variants

Treatment-related:

- Local recurrence, considerably more frequent in atypical/malignant grades despite complete resection
- Radiation-induced complications (cognitive change, radionecrosis) particularly relevant given generally long survival even with higher-grade disease
- Venous sinus injury during surgical resection of parasagittal/falcine tumours
- Cranial nerve deficits from surgery near skull-base locations

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone	Perioperative oedema control	4-8 mg/day in divided doses, tapered post-operatively	Hyperglycaemia, mood disturbance, immunosuppression with prolonged use

★ CLINICAL PEARLS & EXAM FOCUS

► Grade (1 vs 2 vs 3), not just complete surgical excision, determines recurrence risk and the need for adjuvant radiotherapy – histological grading should always guide follow-up planning.

► Many grade 1 meningiomas can be safely observed rather than operated on, particularly incidental, small, asymptomatic lesions in older patients.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Central Nervous System Cancers (current version)

M78 Medulloblastoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How does molecular subgrouping influence medulloblastoma treatment intensity? [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Tailor treatment intensity to molecular risk in paediatric neuro-oncology (CD8, CD2.1)

DEFINITION The commonest malignant paediatric brain tumour, arising in the cerebellum, with a propensity to seed via the cerebrospinal fluid to the spine; molecular subgrouping (WNT, SHH, Group 3, Group 4) now guides risk stratification and is central to modern management.

Stage / Setting	Principal Treatment Approach
Standard-risk disease	Maximal safe resection followed by craniospinal irradiation (reduced dose in standard-risk) and adjuvant chemotherapy
High-risk disease (residual tumour, metastatic at diagnosis, certain molecular subgroups)	Maximal resection followed by full-dose craniospinal irradiation and intensified chemotherapy
Very young children (under 3 years)	Chemotherapy-focused approach to delay or avoid craniospinal irradiation given severe neurocognitive impact on the developing brain

COMPLICATIONS

Disease-related:

- Hydrocephalus from fourth ventricle obstruction, often the presenting complication requiring urgent CSF diversion
- Leptomeningeal/spinal seeding via CSF dissemination, assessed with spinal MRI and CSF cytology at diagnosis
- Posterior fossa syndrome (cerebellar mutism) after surgery – transient but distressing loss of speech with emotional lability
- Cranial nerve deficits from brainstem proximity of the tumour/surgical site

Treatment-related:

- Severe neurocognitive and endocrine (growth, pubertal, thyroid) impairment from craniospinal irradiation, particularly in young children – a major long-term survivorship concern
- Ototoxicity from cisplatin, compounding any radiation-related hearing effects
- Growth hormone deficiency and other hypopituitarism from radiation to the hypothalamic-pituitary axis
- Secondary malignancies as a late effect of craniospinal irradiation

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin + Vincristine + Cyclophosphamide	Adjuvant chemotherapy backbone	Per standard risk-stratified protocol	Ototoxicity/nephrotoxicity (cisplatin), peripheral neuropathy (vincristine), myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

▸ Molecular subgrouping (particularly WNT-activated, generally excellent prognosis) is increasingly used to tailor treatment intensity and avoid over-treatment – an important recent-advances point.

▸ Long-term survivorship care (endocrine, neurocognitive, auditory) is as important as acute treatment given many children becoming long-term survivors.

GUIDELINES & RECENT ADVANCES

- NCCN/SIOP Guidelines: Pediatric Central Nervous System Tumours (current consensus)

M79 Primary CNS Lymphoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why should steroids be withheld before biopsy in suspected primary CNS lymphoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Avoid diagnostic pitfalls from premature corticosteroid use in neuro-oncology (CD2.1, PD3)

DEFINITION An aggressive extranodal non-Hodgkin lymphoma (usually diffuse large B-cell type) confined to the brain, eyes, or spinal cord, with increased incidence in immunosuppressed patients (HIV, transplant recipients) and rising incidence in immunocompetent elderly individuals.

Stage / Setting	Principal Treatment Approach
Immunocompetent, fit for intensive therapy	High-dose methotrexate-based induction chemotherapy (± rituximab) followed by consolidation (autologous stem cell transplantation or reduced-dose whole-brain radiotherapy)
Elderly/frail, unfit for intensive therapy	Lower-intensity methotrexate-based regimens, with careful risk-benefit discussion
HIV-associated disease	Combination antiretroviral therapy alongside chemotherapy, with careful attention to drug interactions

COMPLICATIONS

Disease-related:

- Raised intracranial pressure and focal neurological deficit from tumour mass effect
- Ocular involvement causing visual disturbance (vitreous/uveal infiltration)
- Corticosteroid-induced rapid tumour shrinkage before biopsy, which can obscure histological diagnosis if steroids are given prematurely
- Cognitive decline from both the tumour itself and, historically, whole-brain radiotherapy

Treatment-related:

- Nephrotoxicity from high-dose methotrexate if hydration/alkalinisation/monitoring protocols are not meticulously followed
- Delayed methotrexate clearance causing severe systemic toxicity (mucositis, myelosuppression) if renal function is impaired
- Neurocognitive decline, particularly with combined chemotherapy-whole-brain-radiotherapy approaches, now avoided where possible
- Opportunistic infection risk, particularly in HIV-associated disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
High-dose Methotrexate	Induction chemotherapy	3-8 g/m² IV per protocol, with leucovorin rescue and urine alkalinisation	Nephrotoxicity (requires adequate hydration/alkalinisation), mucositis,
	backbone, intensive approach		neurotoxicity – requires therapeutic drug monitoring
Rituximab	Added to methotrexate-based regimens	375-500 mg/m² IV per protocol schedule	Infusion reactions, hepatitis B reactivation risk

★ CLINICAL PEARLS & EXAM FOCUS

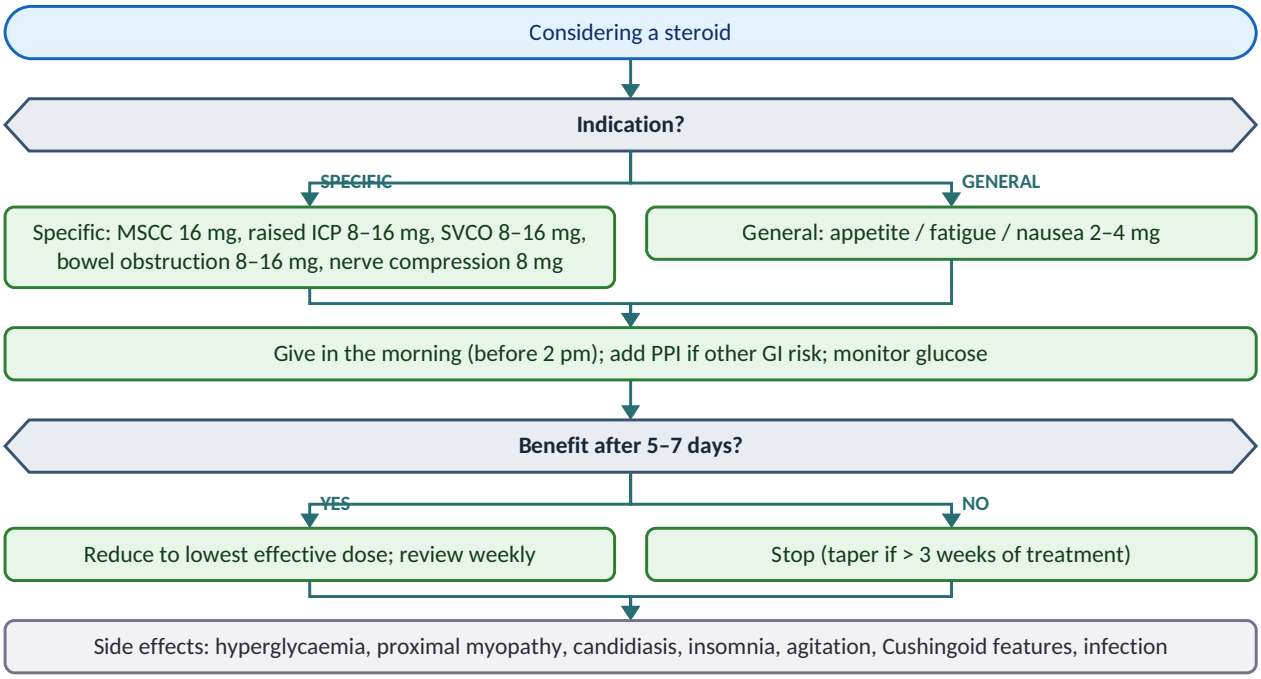
▸ Corticosteroids should be withheld before biopsy whenever clinically safe, since they can rapidly shrink the tumour and cause a non-diagnostic ('ghost tumour') biopsy result.

▸ High-dose methotrexate requires strict protocol adherence (hydration, urine alkalinisation, leucovorin rescue, level monitoring) to prevent severe, sometimes fatal toxicity.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Central Nervous System Cancers (current version)

ALGORITHM · Use of Corticosteroids in Palliative Care Algo 60 — full page ›



★ KEY POINTS

- ▶ Proximal myopathy appears after 2–4 weeks and limits mobility.
- ▶ Steroid-induced hyperglycaemia peaks in the afternoon — check evening glucose.

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M80 Brain Metastases

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is stereotactic radiosurgery often preferred over whole-brain radiotherapy today? [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Balance local tumour control against neurocognitive preservation in brain metastasis management (CD4.1, AD5)

DEFINITION The commonest intracranial tumour in adults overall, arising from systemic malignancy (most often lung, breast, melanoma, renal cell and colorectal cancer), representing a distinct clinical entity requiring specific multidisciplinary management separate from the primary cancer.

Stage / Setting	Principal Treatment Approach
Single/oligometastatic, good performance status	Stereotactic radiosurgery ± surgical resection for larger/symptomatic lesions, with consideration of deferring whole-brain radiotherapy to spare cognition
Multiple metastases, good performance status	Stereotactic radiosurgery to multiple lesions where feasible, or whole-brain radiotherapy for very numerous/diffuse disease
Poor performance status/extensive systemic disease	Best supportive care with corticosteroids for symptom palliation, given limited benefit of aggressive local therapy in this setting

COMPLICATIONS

Disease-related:

- Seizures, a common presenting feature, particularly with cortical lesions
- Raised intracranial pressure causing headache, vomiting and declining consciousness, occasionally progressing to herniation (a neurological emergency)
- Haemorrhage into metastases, particularly common with melanoma, renal cell carcinoma, choriocarcinoma and thyroid cancer
- Leptomeningeal spread, a diffuse and generally poor-prognosis pattern of CNS involvement

Treatment-related:

- Steroid-related complications (hyperglycaemia, myopathy, psychiatric disturbance) from prolonged dexamethasone use
- Radiation necrosis, a delayed complication of stereotactic radiosurgery that can mimic tumour progression on imaging
- Neurocognitive decline from whole-brain radiotherapy, prompting increasing preference for stereotactic approaches where feasible
- Surgical complications (infection, haemorrhage, neurological deficit) after resection of symptomatic lesions

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone			
	Symptom control for oedema-related mass effect	4-16 mg/day in divided doses, tapered as tolerated	Hyperglycaemia, myopathy, mood disturbance, immunosuppression
Levetiracetam	Seizure prophylaxis/ treatment if seizures have occurred	500-1500 mg orally twice daily	Mood/behavioural disturbance; routine prophylaxis in seizure-naive patients is not recommended

★ CLINICAL PEARLS & EXAM FOCUS

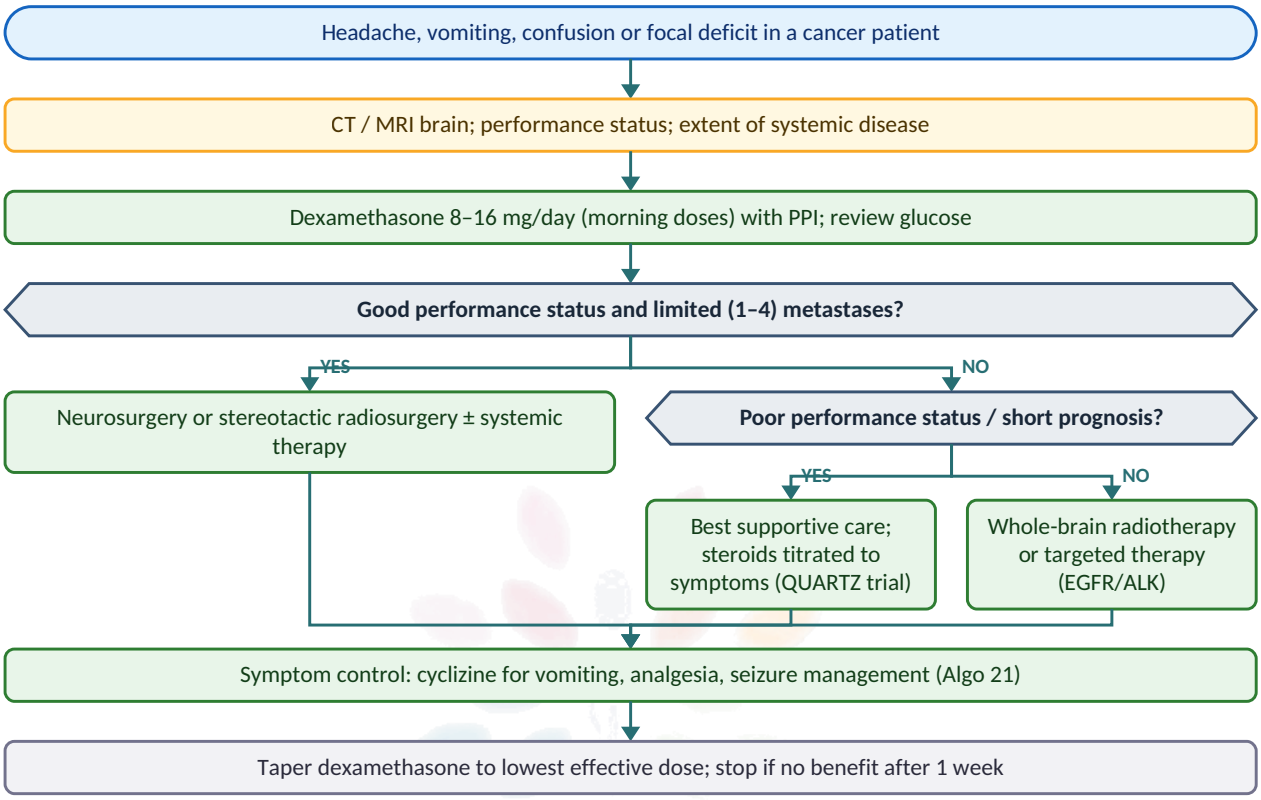
▸ Stereotactic radiosurgery with deferred whole-brain radiotherapy is now preferred where feasible, specifically to spare neurocognitive function – an important recent-advances/quality-of-life point.

▸ Routine prophylactic antiepileptic use in seizure-naive brain metastasis patients is not recommended and should be reserved for those who have actually had a seizure.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Central Nervous System Cancers (current version)

ALGORITHM · Raised Intracranial Pressure and Brain Metastases Algo 22 — full page ›



★ KEY POINTS

▸ QUARTZ: WBRT added little to quality-adjusted survival in poor-prognosis NSCLC brain metastases.

▸ Give steroids before 2 pm to reduce insomnia.

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M81 Pituitary Adenoma / Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the emergency management of pituitary apoplexy. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and urgently manage neuro-endocrine emergencies (CD4.1, PD3)

DEFINITION Pituitary adenomas are common, usually benign tumours of the anterior pituitary, which may be functioning (secreting prolactin, growth hormone, ACTH) causing distinct endocrine syndromes, or non-functioning, presenting with mass effect; true pituitary carcinoma (with metastasis) is exceedingly rare.

Stage / Setting	Principal Treatment Approach
Prolactinoma (functioning)	Dopamine agonist therapy (cabergoline) as first-line, often avoiding surgery entirely given excellent medical response
Other functioning adenomas (growth hormone/ACTH-secreting) or non-functioning with mass effect	Transsphenoidal surgical resection as first-line, with medical therapy as adjunct/alternative
Residual/recurrent disease, or rare pituitary carcinoma	Radiotherapy for residual disease; temozolomide chemotherapy for aggressive/carcinoma cases

COMPLICATIONS

Disease-related:

- Visual field defect (classically bitemporal hemianopia) from optic chiasm compression by a growing macroadenoma
- Hypopituitarism from compression of normal pituitary tissue, causing secondary adrenal insufficiency, hypothyroidism, or hypogonadism
- Pituitary apoplexy – sudden haemorrhage/infarction into the adenoma causing severe headache, visual loss and acute adrenal crisis, a neuro-endocrine emergency
- Cushing's disease (ACTH-secreting adenoma) causing metabolic, cardiovascular and infective complications of hypercortisolism

Treatment-related:

- CSF rhinorrhoea and risk of meningitis after transsphenoidal surgery
- Permanent hypopituitarism requiring lifelong hormone replacement after surgery/radiotherapy
- Cardiac valvulopathy, a rare but recognised risk with very high cumulative cabergoline doses
- Visual deterioration if apoplexy or mass effect is not promptly recognised and treated

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cabergoline	First-line therapy for prolactinoma	0.25-1 mg orally twice weekly, titrated to prolactin level	Nausea, orthostatic hypotension, rare cardiac valvulopathy with very high cumulative doses
Octreotide LAR	Growth hormone-secreting adenoma (acromegaly), adjunct/ alternative to surgery	20-30 mg IM every 4 weeks	Gallstones, hyperglycaemia, GI discomfort

★ CLINICAL PEARLS & EXAM FOCUS

► Pituitary apoplexy is a neuro-endocrine emergency requiring urgent corticosteroid (stress-dose hydrocortisone) administration even before definitive imaging/surgical decision-making, given risk of fatal adrenal crisis.

► Dopamine agonist therapy alone resolves the majority of prolactinomas without needing surgery – an important distinction from other pituitary tumour types.

GUIDELINES & RECENT ADVANCES

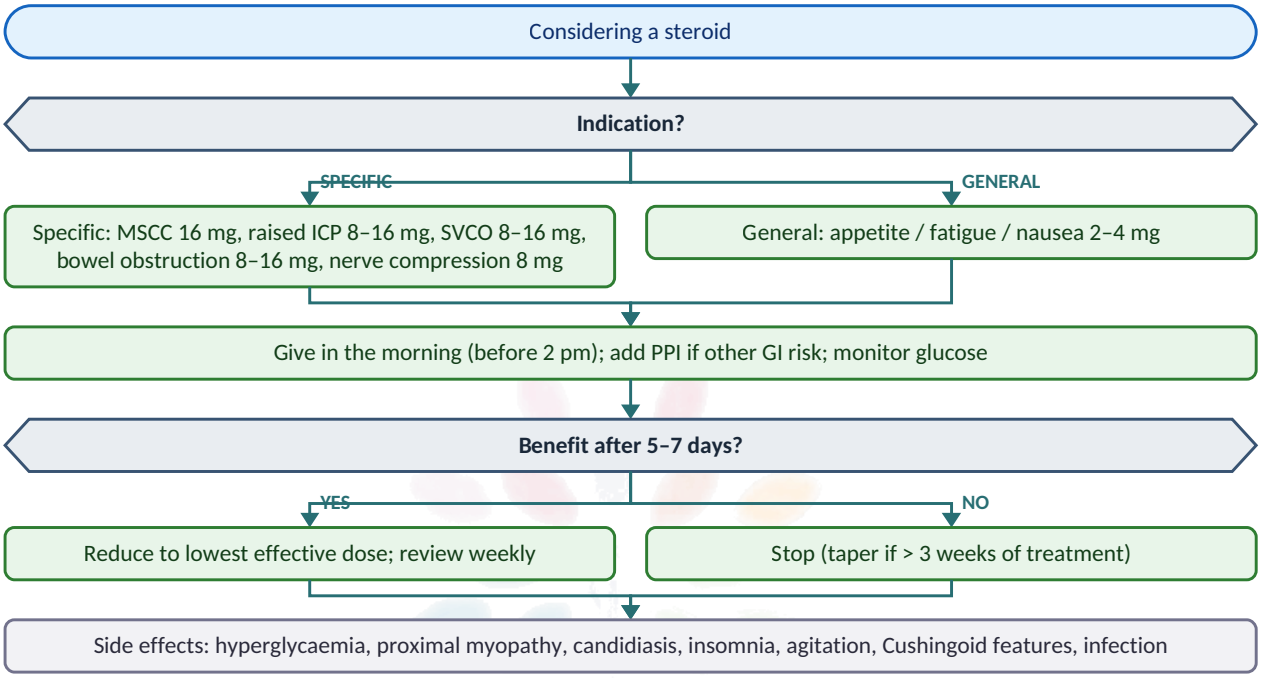
- Pituitary Society / Endocrine Society Clinical Practice Guidelines

EXAM-ORIENTED THEORY

Palliative care focus — pituitary tumours

Problem	Management
Headache, visual loss	Analgesia, steroids, surgery / radiotherapy
Hypopituitarism	Hydrocortisone (sick-day rules), thyroxine; never stop steroids abruptly
Diabetes insipidus	Desmopressin; monitor sodium
Pituitary apoplexy	Emergency hydrocortisone 100 mg IV; neurosurgery

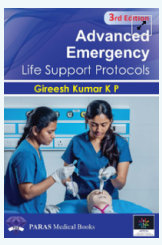
ALGORITHM · Use of Corticosteroids in Palliative Care Algo 60 — full page ›



★ KEY POINTS

- ▸ Proximal myopathy appears after 2–4 weeks and limits mobility.
- ▸ Steroid-induced hyperglycaemia peaks in the afternoon — check evening glucose.

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M82 Osteosarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is persistent adolescent bone pain a red flag requiring imaging? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise bone malignancy presenting as musculoskeletal pain in young patients (CD4.1, CD8)

DEFINITION The commonest primary malignant bone tumour, typically affecting the metaphysis of long bones (distal femur, proximal tibia) in adolescents and young adults, presenting with localised bone pain and swelling, often initially attributed to injury or growing pains.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Neoadjuvant chemotherapy followed by limb-salvage or amputation surgery, then adjuvant chemotherapy based on histological response
Poor histological response to neoadjuvant chemotherapy	Intensified/alternative adjuvant chemotherapy regimens, though prognostic benefit is limited
Metastatic (commonly lung) at diagnosis	Chemotherapy combined with surgical resection of both primary and resectable pulmonary metastases where feasible

COMPLICATIONS

Disease-related:

- Pathological fracture through the weakened bone
- Pulmonary metastases, the dominant and often sole site of distant spread
- Local recurrence after limb-salvage surgery, occasionally necessitating later amputation
- Severe bone pain, often the presenting and persisting symptom despite analgesia

Treatment-related:

- Cardiotoxicity from cumulative anthracycline dose, requiring baseline and serial cardiac function monitoring
- Nephrotoxicity from cisplatin and high-dose methotrexate, requiring meticulous hydration/monitoring protocols
- Limb-salvage surgical complications (infection, prosthesis failure, limb-length discrepancy in growing children)
- Ototoxicity from cisplatin, particularly relevant given young patient age and lifelong impact

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin + Doxorubicin + High-dose Methotrexate (MAP)	Standard chemotherapy backbone	Per standard multi-agent protocol	Nephro/ototoxicity (cisplatin), cardiotoxicity (doxorubicin), mucositis/
over several			nephrotoxicity
months			(methotrexate, requires
			leucovorin rescue)

★ CLINICAL PEARLS & EXAM FOCUS

► Persistent, worsening bone pain in an adolescent, especially with overlying swelling or a palpable mass, should never be dismissed as simple 'growing pains' without imaging.

► Degree of histological necrosis after neoadjuvant chemotherapy is the single most important prognostic factor and guides further treatment intensity.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Bone Cancer (current version)

M83 Ewing Sarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must mesna be co-administered with ifosfamide? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Prevent chemotherapy-specific uro-toxicity in sarcoma treatment (CD2.1, PD3)

DEFINITION An aggressive small round blue cell tumour of bone or soft tissue, driven by the EWSR1-FLI1 gene fusion, typically affecting the diaphysis of long bones or the pelvis in children and young adults; presents with pain, swelling, and sometimes systemic symptoms (fever, raised inflammatory markers) mimicking infection.

Stage / Setting	Principal Treatment Approach
Localised	Neoadjuvant chemotherapy followed by local control (surgery and/or radiotherapy) and further adjuvant chemotherapy
Metastatic at diagnosis (lung, bone, bone marrow)	Intensive chemotherapy with local control of the primary site; whole-lung irradiation considered for pulmonary metastases
Relapsed/refractory disease	Alternative chemotherapy combinations, with generally poor outcomes once relapse occurs

COMPLICATIONS

Disease-related:

- Pathological fracture through the bone lesion
- Pulmonary and bone marrow metastases, important determinants of prognosis
- Local complications from pelvic primaries, including nerve/vascular compression given deep anatomical location
- Systemic inflammatory symptoms (fever, weight loss) that can mimic osteomyelitis and delay diagnosis

Treatment-related:

- Haemorrhagic cystitis from ifosfamide/cyclophosphamide if mesna prophylaxis and hydration are inadequate
- Cardiotoxicity from cumulative anthracycline dose
- Infertility, particularly relevant given young patient age, from alkylating agent exposure
- Secondary malignancy (leukaemia) as a rare but serious late effect of etoposide-containing regimens

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication
Vincristine + Doxorubicin + Cyclophosphamide, alternating with Ifosfamide + Etoposide (VDC/IE)	Standard chemotherapy backbone

DOSE

Per standard alternati multi-agent protocol

★ CLINICAL PEARLS & EXAM FOCUS

► Fever and raised inflammatory markers alongside bone pain in a young patient can mimic osteomyelitis – imaging and biopsy should not be delayed if symptoms persist despite antibiotic trial.

► Mesna must always accompany ifosfamide/high-dose cyclophosphamide to prevent haemorrhagic cystitis – a frequently tested pharmacology safety point.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Bone Cancer (current version)

AETCM Academy for Emergency

M84 Chondrosarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is chondrosarcoma managed differently from osteosarcoma in terms of chemotherapy use? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise differential chemosensitivity across bone sarcoma subtypes (CD2.1, CD4.1)

DEFINITION A malignant cartilage-forming tumour, typically affecting the pelvis, proximal femur or proximal humerus in middle-aged to older adults, generally slower-growing and less chemosensitive than osteosarcoma or Ewing sarcoma, making complete surgical excision the primary treatment modality.

Stage / Setting	Principal Treatment Approach
Low-grade, localised	Wide surgical excision (curettage alone is inadequate given recurrence risk, even for low-grade disease)
High-grade or dedifferentiated	Wide surgical excision, with chemotherapy considered for the dedifferentiated (high-grade) component specifically
Metastatic/unresectable	Limited systemic therapy options given relative chemo- and radio-resistance; best supportive/ palliative care often predominates

COMPLICATIONS

Disease-related:

- Local recurrence if excision margins are inadequate, given cartilage's resistance to simple curettage
- Pelvic chondrosarcoma causing nerve/vascular compression given deep, complex anatomical location
- Dedifferentiation to a high-grade, more aggressive component within an otherwise low-grade tumour
- Pulmonary metastases in high-grade/dedifferentiated disease

Treatment-related:

- Major surgical morbidity from wide excision of pelvic/complex-site tumours, sometimes requiring extensive reconstruction
- Limited systemic therapy options for metastatic conventional chondrosarcoma, given marked chemo-/ radio-resistance, concentrating management on symptom-directed palliative care
- Nerve injury from surgery near major pelvic/limb neurovascular structures
- Functional limb/pelvic girdle impairment after extensive resection

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Doxorubicin-based chemotherapy	Dedifferentiated chondrosarcoma only (not conventional chondrosarcoma, which is chemoresistant)	Per standard sarcoma chemotherapy protocol	Cardiotoxicity, myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

► Conventional chondrosarcoma is notably chemoresistant – this distinguishes it sharply from osteosarcoma/Ewing sarcoma and should guide realistic treatment expectation discussions.

► Adequate wide surgical margins, not curettage, are essential even for low-grade chondrosarcoma given its tendency to recur locally.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Bone Cancer (current version)

M85 Leiomyosarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What clinical feature should raise suspicion of leiomyosarcoma over a benign fibroid? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Distinguish malignant from benign smooth muscle tumours on clinical grounds (CD4.1)

DEFINITION A malignant tumour of smooth muscle origin, arising most commonly in the uterus, retroperitoneum, or deep soft tissue of the extremities, often presenting as a painless, enlarging mass or (in uterine disease) as atypical vaginal bleeding/a rapidly growing fibroid.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Wide surgical excision with clear margins, the primary curative approach
High-risk resected disease	Adjuvant chemotherapy or radiotherapy considered based on size, grade and margin status, though evidence for survival benefit is limited
Metastatic/ unresectable	Doxorubicin-based chemotherapy first-line, with gemcitabine-docetaxel or other agents on progression

COMPLICATIONS

Disease-related:

- Pulmonary metastases, the commonest site of distant spread, sometimes multiple and bilateral
- Rapid local growth causing mass effect symptoms depending on site (pelvic pressure, bowel/bladder compression with retroperitoneal/uterine tumours)
- Haemorrhage from a necrotic, friable tumour mass
- Local recurrence despite apparently adequate excision, given often infiltrative microscopic margins

Treatment-related:

- Cardiotoxicity from cumulative anthracycline exposure, limiting total lifetime chemotherapy dose
- Surgical morbidity from resection of large retroperitoneal/pelvic tumours (bowel, vascular, ureteric injury)
- Pneumonitis, rarely, with gemcitabine-docetaxel combination therapy
- Significant symptom burden from pulmonary metastases (breathlessness, haemoptysis) in advanced disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Doxorubicin	First-line metastatic chemotherapy	60-75 mg/m² IV every 3 weeks (or dose-dense schedules)	Cardiotoxicity – requires baseline and cumulative-dose cardiac monitoring
Gemcitabine + Docetaxel	Second-line or first-line alternative	Gemcitabine 900 mg/m² days 1,8 + docetaxel 75-100 mg/m² day 8, every 21 days	Myelosuppression, peripheral neuropathy, pneumonitis (rare)

★ CLINICAL PEARLS & EXAM FOCUS

► A rapidly enlarging uterine mass in a peri-/post-menopausal woman should raise suspicion of leiomyosarcoma rather than being assumed to be a benign fibroid.

► Leiomyosarcoma has a strong predilection for pulmonary metastasis – surveillance imaging typically focuses heavily on chest imaging.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Soft Tissue Sarcoma (current version)

M86 Liposarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How does liposarcoma subtype influence treatment and prognosis? [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply histological subtyping to individualised sarcoma management (CD2.1, CD4.1)

DEFINITION A malignant tumour of adipocytic origin and the commonest soft tissue sarcoma overall, ranging from well-differentiated (locally aggressive but low metastatic potential) to dedifferentiated, myxoid, and pleomorphic subtypes with progressively more aggressive behaviour; typically arises in the retroperitoneum or deep thigh.

Stage / Setting	Principal Treatment Approach
Well-differentiated, localised	Wide surgical excision; recurrence is managed with repeat resection given low metastatic risk of this subtype
Myxoid/round-cell or dedifferentiated, localised	Wide surgical excision ± (neo)adjuvant radiotherapy; myxoid liposarcoma is notably radiosensitive
Metastatic/unresectable, pleomorphic or dedifferentiated	Doxorubicin-based chemotherapy, with trabectedin as an option particularly in myxoid/round-cell subtype

COMPLICATIONS

Disease-related:

- Local recurrence, particularly frequent and often multiple with well-differentiated retroperitoneal disease given difficulty achieving complete excision in this location
- Progressive mass effect on retroperitoneal structures (bowel, ureters, major vessels) from large tumours
- Dedifferentiation to a higher-grade component within a previously well-differentiated tumour
- Distant metastasis (lung), primarily a concern with dedifferentiated, myxoid/round-cell, and pleomorphic subtypes rather than well-differentiated disease

Treatment-related:

- Major surgical morbidity from resection of large retroperitoneal masses, often requiring multivisceral resection
- Cardiotoxicity from cumulative anthracycline exposure
- Haemorrhagic cystitis from ifosfamide if mesna cover is inadequate
- Multiple repeat surgeries over time for recurrent well-differentiated disease, with cumulative surgical morbidity

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Doxorubicin + Ifosfamide	Metastatic/high-risk disease chemotherapy	Per standard combination sarcoma protocol	Cardiotoxicity, haemorrhagic cystitis (requires mesna), myelosuppression
Trabectedin	Relapsed/refractory disease, particularly myxoid/round-cell subtype	1.5 mg/m² IV over 24 hours, every 3 weeks	Hepatotoxicity, myelosuppression, rhabdomyolysis (rare)

★ CLINICAL PEARLS & EXAM FOCUS

► Histological subtype (well-differentiated vs myxoid vs dedifferentiated vs pleomorphic) fundamentally changes both prognosis and treatment approach – liposarcoma is not a single clinical entity.

► Myxoid liposarcoma is distinctively radiosensitive and has an unusual metastatic pattern (can spread to unusual soft tissue/bone sites) worth remembering for exams.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Soft Tissue Sarcoma (current version)

M87 Synovial Sarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What general principle of soft tissue mass evaluation does synovial sarcoma illustrate? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply pre-biopsy imaging principles before excising a suspicious soft tissue mass (CD4.1, PD3)

DEFINITION A translocation-associated (SYT-SSX fusion) soft tissue sarcoma, despite its name arising most often near but not within joints, typically affecting the extremities of adolescents and young adults as a slow-growing mass that can be mistaken for a benign lesion given its deceptively indolent appearance.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Wide surgical excision ± adjuvant radiotherapy, particularly for larger/high-grade tumours
Large, high-risk localised disease	Neoadjuvant chemotherapy considered given relative chemosensitivity compared with other soft tissue sarcomas
Metastatic/unresectable	Ifosfamide-based chemotherapy, to which this sarcoma subtype is notably more responsive than most soft tissue sarcomas

COMPLICATIONS

Disease-related:

- Pulmonary metastases, the dominant site of distant spread
- Local recurrence, particularly if initial excision was performed assuming a benign diagnosis (inadequate margins)
- Deceptively slow growth leading to diagnostic delay, sometimes years, before malignancy is suspected
- Lymph node metastasis, somewhat more frequent than in most other soft tissue sarcomas

Treatment-related:

- Ifosfamide-induced encephalopathy, an uncommon but important neurotoxicity requiring prompt recognition and methylene blue treatment if severe
- Haemorrhagic cystitis without adequate mesna prophylaxis
- Cardiotoxicity from cumulative anthracycline exposure
- Positive margins and local recurrence if the lesion was initially excised without pre-operative imaging/ biopsy under a mistaken benign diagnosis

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Ifosfamide + Doxorubicin	First-line chemotherapy, given relative chemosensitivity	Per standard combination sarcoma protocol with mesna cover	Haemorrhagic cystitis (requires mesna), cardiotoxicity, myelosuppression, CNS toxicity (ifosfamide encephalopathy, uncommon)
Pazopanib			
Relapsed/refractory disease		800 mg orally once daily	Hypertension, hepatotoxicity, impaired wound healing

★ CLINICAL PEARLS & EXAM FOCUS

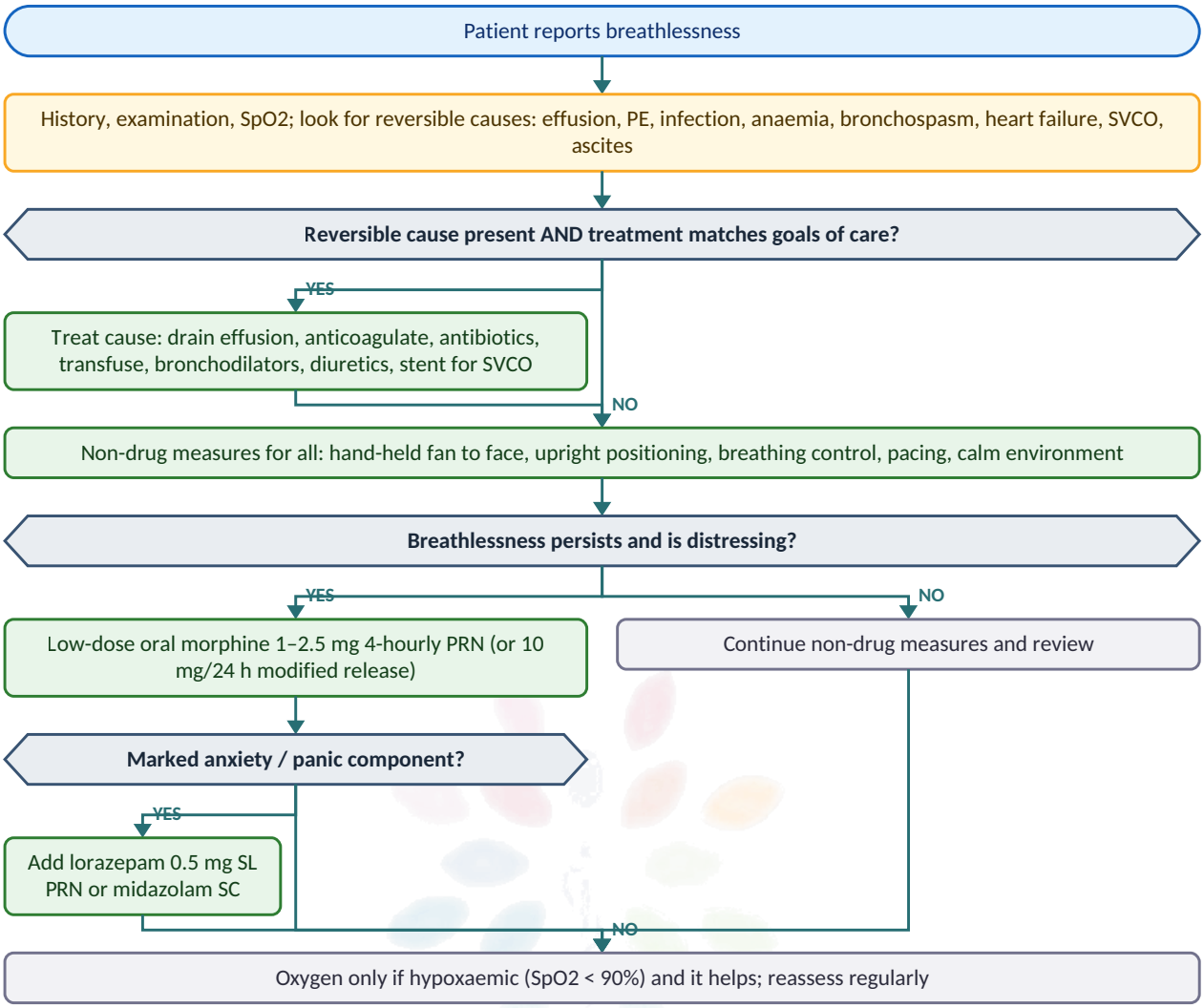
▸ Any soft tissue mass, however slow-growing or 'benign-appearing', arising deep to fascia or larger than 5 cm should be imaged and biopsied before excision – a core sarcoma-management principle illustrated well by this tumour.

▸ Synovial sarcoma is relatively chemosensitive compared with most adult soft tissue sarcomas, an important exception worth remembering.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Soft Tissue Sarcoma (current version)

ALGORITHM · Approach to Breathlessness (Dyspnoea) in Palliative Care Algo 5 — full page ›



★ KEY POINTS

- ▸ Opioids reduce the sensation of breathlessness without clinically significant respiratory depression at low doses.
- ▸ The fan directed at the face (trigeminal stimulation) is evidence-based and costless.

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M88 Rhabdomyosarcoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why are parameningeal sites of particular concern in rhabdomyosarcoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise anatomical risk for intracranial extension in paediatric sarcoma (CD8, CD4.1)

DEFINITION The commonest soft tissue sarcoma of childhood, arising from primitive skeletal muscle precursor cells, occurring at varied sites (head/neck, genitourinary tract, extremities) with embryonal and alveolar histological subtypes carrying different prognoses.

Stage / Setting	Principal Treatment Approach
Localised, favourable site/histology	Multi-agent chemotherapy combined with surgery and/or radiotherapy for local control
Localised, unfavourable site/histology (alveolar subtype)	More intensive multi-agent chemotherapy with aggressive local control
Metastatic disease	Intensive multi-agent chemotherapy, though outcomes remain poor in metastatic alveolar subtype particularly

COMPLICATIONS

Disease-related:

- Site-specific complications – bladder/prostate tumours causing urinary obstruction, orbital tumours causing proptosis/visual compromise, parameningeal tumours (nasopharynx/sinuses) risking direct intracranial extension
- Lymph node and distant metastasis (lung, bone, bone marrow), particularly in alveolar subtype
- Local recurrence, especially with incomplete initial resection or unfavourable histology
- Functional/cosmetic deficit from the primary site itself (e.g. orbital, genitourinary) even with successful tumour control

Treatment-related:

- Haemorrhagic cystitis from cyclophosphamide without adequate mesna/hydration
- Pelvic/genitourinary functional complications (urinary, sexual, fertility) after treatment of bladder/ prostate/ vaginal primary sites
- Growth and endocrine effects of radiotherapy in growing children, particularly with head/neck or pelvic fields
- Psychosocial impact on the child and family given the intensive, prolonged multi-modality treatment course

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Vincristine + Actinomycin-D + Cyclophosphamide (VAC)		Per standard paediatric	Myelosuppression, vincristine neuropathy/

Standard	protocol,	constipation,
chemotherapy	risk-	haemorrhagic cystitis
backbone	stratified	with cyclophosphamide
	duration	(requires mesna/
		hydration)

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★ CLINICAL PEARLS & EXAM FOCUS

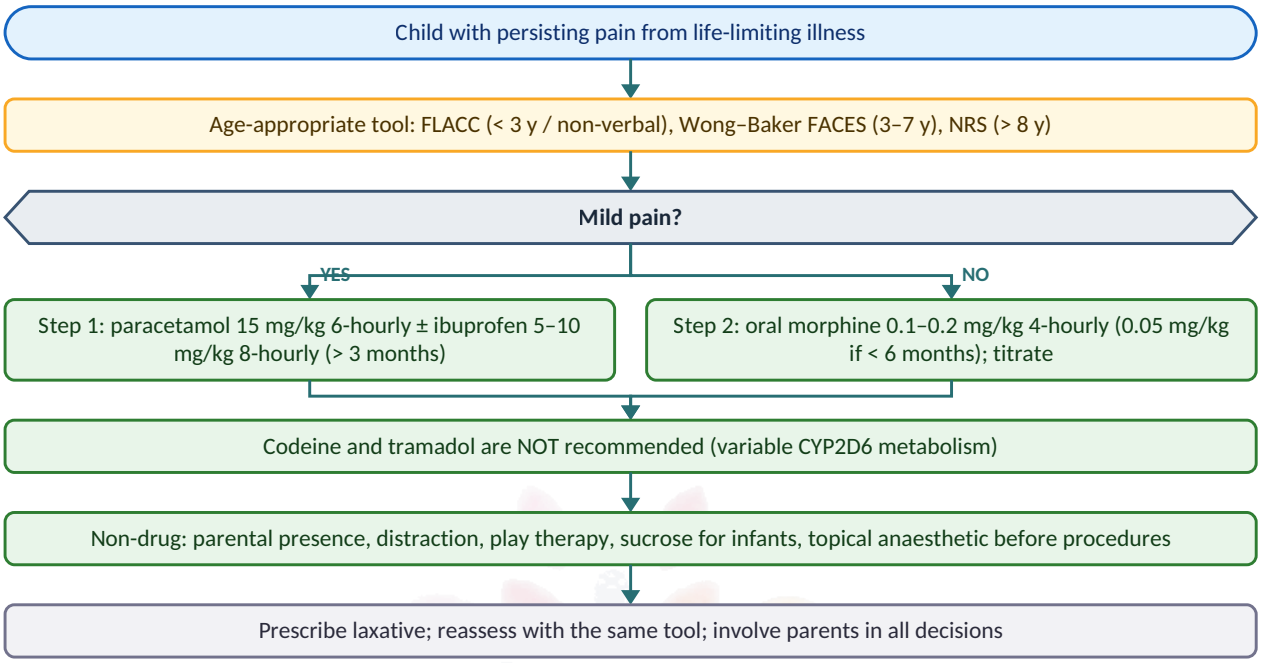
▸ Parameningeal rhabdomyosarcoma (nasopharynx, sinuses, middle ear) carries particular risk of direct intracranial extension and should prompt careful skull-base imaging.

▸ Histological subtype (embryonal vs alveolar) is a key prognostic factor independent of site and stage, and should always be specifically noted in management discussions.

GUIDELINES & RECENT ADVANCES

- NCCN/COG Guidelines: Pediatric Rhabdomyosarcoma (current consensus)

ALGORITHM · Pain Management in Children Algo 32 — full page ›

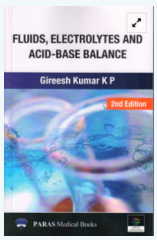


★ KEY POINTS

▸ Children are not small adults — dose by weight and age.

▸ ACT categories 1–4 classify life-limiting conditions for paediatric palliative care.

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Dr. Gireesh Kumar K P · Principal, Amrita School of Medicine · Director, AETCM, Kochi

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M89 Desmoid-Type Fibromatosis

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why has 'wait and see' become first-line management for desmoid tumours? [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply evidence-based observation-first strategies in locally aggressive, non-metastasising tumours (CD4.1, AD3)

DEFINITION A locally aggressive, clonal fibroblastic proliferation that does not metastasise but can cause substantial morbidity through relentless local infiltration; associated with beta-catenin pathway mutations sporadically or with APC mutations in familial adenomatous polyposis (Gardner syndrome).

Stage / Setting	Principal Treatment Approach
Asymptomatic or minimally symptomatic, stable disease	Active surveillance/observation, as spontaneous stabilisation or even regression occurs in a substantial proportion of cases
Symptomatic/progressive disease	Systemic therapy – anti-oestrogen agents (tamoxifen), NSAIDs, or tyrosine kinase inhibitors (sorafenib) as first-line, reserving surgery for failures given high local recurrence rates
Surgery/radiotherapy	Reserved for disease refractory to systemic therapy or causing critical structure compromise, given recognised high recurrence rates after surgery alone

COMPLICATIONS

Disease-related:

- Progressive local infiltration causing pain, functional limitation, or compression of adjacent nerves/ vessels/ bowel depending on site
- Intra-abdominal/mesenteric desmoids (particularly in FAP/Gardner syndrome) causing bowel obstruction or ischaemia from mesenteric vessel encasement
- High local recurrence rate after surgical resection alone, historically the primary driver of a shift toward systemic/observational management
- Significant psychological distress from a tumour that is 'not cancer' yet can be profoundly disfiguring/ disabling

Treatment-related:

- Surgical morbidity disproportionate to the tumour's non-metastasising nature, given the need for wide margins near critical structures
- Recurrence despite surgery, sometimes multiple times, previously leading to escalating, morbid re-operations now largely avoided with modern observation-first approaches
- Hand-foot skin reaction and hypertension limiting tolerability of tyrosine kinase inhibitor therapy
- Bowel ischaemia/infarction from mesenteric vessel encasement in intra-abdominal disease, a serious complication

Drugs used — Drug · Indication · Dose · Caution

Sorafenib	Progressive/symptomatic disease refractory to first-line measures	400 mg orally once daily (dose-reduced per tolerability)	Hand-foot skin reaction, hypertension, diarrhoea, fatigue
Tamoxifen	First-line systemic therapy, lower-intensity option	20 mg orally once daily	Venous thromboembolism, hot flushes; generally well-tolerated compared with chemotherapy

★ CLINICAL PEARLS & EXAM FOCUS

▸ The modern paradigm has shifted decisively toward 'wait and see' as first-line management for desmoid tumours, reflecting recognition of a high spontaneous stabilisation/regression rate – an important recent-advances point often contrary to older teaching.

▸ Desmoid tumours in a patient with colonic polyposis should prompt screening for familial adenomatous polyposis (Gardner syndrome).

GUIDELINES & RECENT ADVANCES

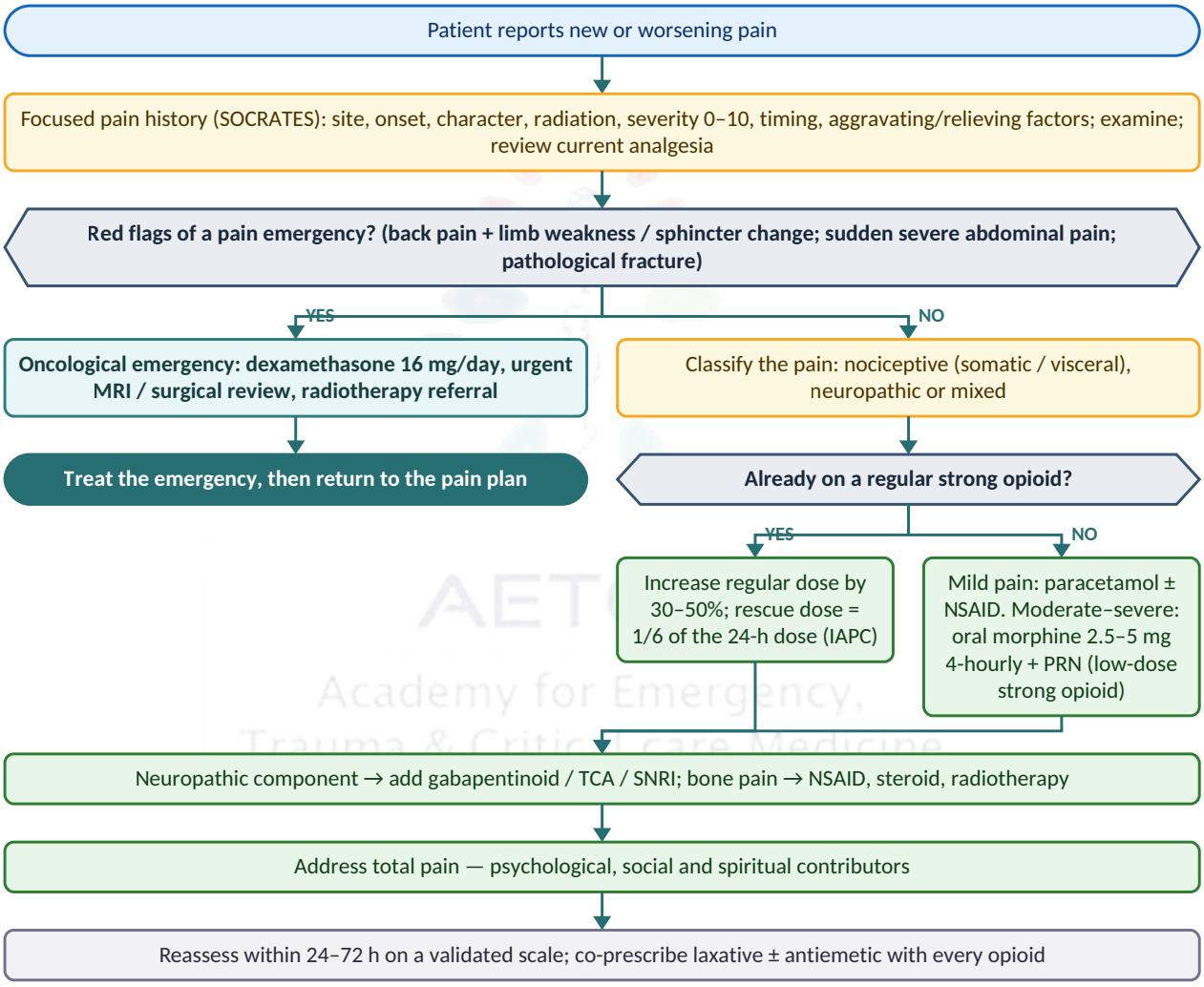
- NCCN Guidelines: Soft Tissue Sarcoma (Desmoid Tumors section, current version)

EXAM-ORIENTED THEORY

Palliative care focus — desmoid tumours

Problem	Management
Pain from locally aggressive mass	NSAIDs (also antitumour effect), adjuvants, opioids
Bowel / ureteric involvement	Stenting, surgery
Progression	Active surveillance first; sorafenib / nirogacestat; low-dose chemo
Body image (abdominal wall)	Psychological support

ALGORITHM · Approach to New or Worsening Pain in a Palliative Care Patient Algo 1 — full page ›



★ KEY POINTS

▸ Always exclude a pain emergency first — spinal cord compression and pathological fracture are time-critical.

▸ Titrate by 30–50% steps; 'by the clock, by the mouth, by the ladder, for the individual'.

M90 Gastrointestinal Neuroendocrine Tumour (Midgut Carcinoid)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is carcinoid heart disease and how does it arise? [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Recognise and screen for serotonin-mediated cardiac complications in neuroendocrine tumours (CD4.1, CD9.1)

DEFINITION Arises from enterochromaffin cells, most often in the small bowel (midgut), frequently indolent and slow-growing but capable of causing carcinoid syndrome (flushing, diarrhoea, bronchospasm, right-sided valvular heart disease) once liver metastases bypass first-pass hepatic hormone clearance.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Surgical resection of the primary tumour with mesenteric lymphadenectomy
Metastatic, liver-dominant, low-grade	Somatostatin analogues for both symptomatic and antiproliferative (disease-control) benefit
Progressive disease despite somatostatin analogues	Peptide receptor radionuclide therapy (PRRT) for somatostatin-receptor-positive disease, or everolimus

COMPLICATIONS

Disease-related:

- Carcinoid syndrome (flushing, secretory diarrhoea, bronchospasm) once liver metastases are present
- Carcinoid heart disease – right-sided valvular fibrosis (tricuspid regurgitation, pulmonary stenosis) from chronic serotonin exposure
- Mesenteric fibrosis causing bowel ischaemia/obstruction, a distinctive midgut carcinoid complication
- Carcinoid crisis (severe hypotension, bronchospasm, flushing) precipitated by anaesthesia, biopsy or tumour manipulation without adequate octreotide cover

Treatment-related:

- Carcinoid crisis during surgery/biopsy without pre-emptive octreotide infusion
- Renal toxicity from PRRT if amino acid nephroprotection protocol is not followed
- Cholelithiasis with long-term somatostatin analogue therapy
- Progressive right heart failure from carcinoid heart disease if unrecognised and untreated

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Octreotide LAR	First-line symptom and disease control	20-30 mg IM every 4 weeks	Gallstones, hyperglycaemia/ hypoglycaemia, injection-site reactions
Lutetium-177 DOTATATE (PRRT)	Progressive, somatostatin-receptor-positive disease	Per approved fixed-interval infusion protocol (typically 4 cycles)	Nephrotoxicity (requires amino acid renal protection), myelosuppression, rare secondary MDS/leukaemia

★ CLINICAL PEARLS & EXAM FOCUS

► All patients with known/suspected functioning carcinoid should receive peri-procedural octreotide cover before any anaesthesia, biopsy, or surgical manipulation to prevent carcinoid crisis.

► Echocardiography should be performed to screen for carcinoid heart disease in patients with long-standing carcinoid syndrome, as valve disease is a major source of morbidity/mortality.

GUIDELINES & RECENT ADVANCES

- NANETS/ENETS Consensus Guidelines: Gastroenteropancreatic Neuroendocrine Tumours

M91 Adrenocortical Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is mitotane therapy accompanied by hormone replacement? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Understand adrenolytic drug mechanism and necessary hormone replacement (CD2.1, CD4.1)

DEFINITION A rare, aggressive malignancy of the adrenal cortex, often functioning (secreting cortisol, androgens, or aldosterone, causing Cushing syndrome, virilisation, or hypertension respectively), typically presenting with a combination of hormonal syndrome and/or a large abdominal mass.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Complete surgical resection (open adrenalectomy preferred over laparoscopic given risk of tumour rupture/seeding); adjuvant mitotane considered for high-risk disease
Locally advanced/ unresectable	Mitotane ± chemotherapy (etoposide-doxorubicin-cisplatin, EDP regimen) combined with mitotane
Metastatic	EDP-mitotane combination chemotherapy as standard first-line systemic therapy

COMPLICATIONS

Disease-related:

- Severe Cushing syndrome complications (hyperglycaemia, hypertension, proximal myopathy, increased infection susceptibility) from cortisol-secreting tumours
- Virilisation/feminisation from androgen/oestrogen-secreting tumours, causing significant psychosocial distress
- Tumour rupture or inferior vena cava tumour thrombus, a surgical emergency
- Rapid local recurrence and metastasis (liver, lung) even after apparently complete resection

Treatment-related:

- Adrenal insufficiency requiring careful glucocorticoid/mineralocorticoid replacement during mitotane therapy, given its adrenolytic mechanism destroying both tumour and residual normal adrenal tissue
- Mitotane-related neurological toxicity (ataxia, confusion) at high blood levels, requiring therapeutic drug monitoring
- Cardiotoxicity and nephrotoxicity from EDP chemotherapy
- Wound/surgical complications from resection of large, vascular adrenal masses

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Mitotane	Adjuvant and advanced/ metastatic disease, adrenolytic agent	Titrated slowly to therapeutic blood levels, typically 2-6 g/day in divided doses	Adrenal insufficiency (requires glucocorticoid/ mineralocorticoid replacement), GI upset,
			neurological toxicity (ataxia, lethargy) at high levels
Etoposide + Doxorubicin + Cisplatin (EDP)	Combined with mitotane, advanced/ metastatic disease	Per standard combination protocol	Myelosuppression, cardiotoxicity, nephrotoxicity

★ CLINICAL PEARLS & EXAM FOCUS

▸ Mitotane requires careful glucocorticoid/mineralocorticoid replacement planning and therapeutic drug level monitoring – it is pharmacologically unlike most other oncology drugs and frequently misunderstood.

▸ Open (not laparoscopic) adrenalectomy is generally preferred for suspected adrenocortical carcinoma to minimise capsular rupture and peritoneal tumour seeding.

GUIDELINES & RECENT ADVANCES

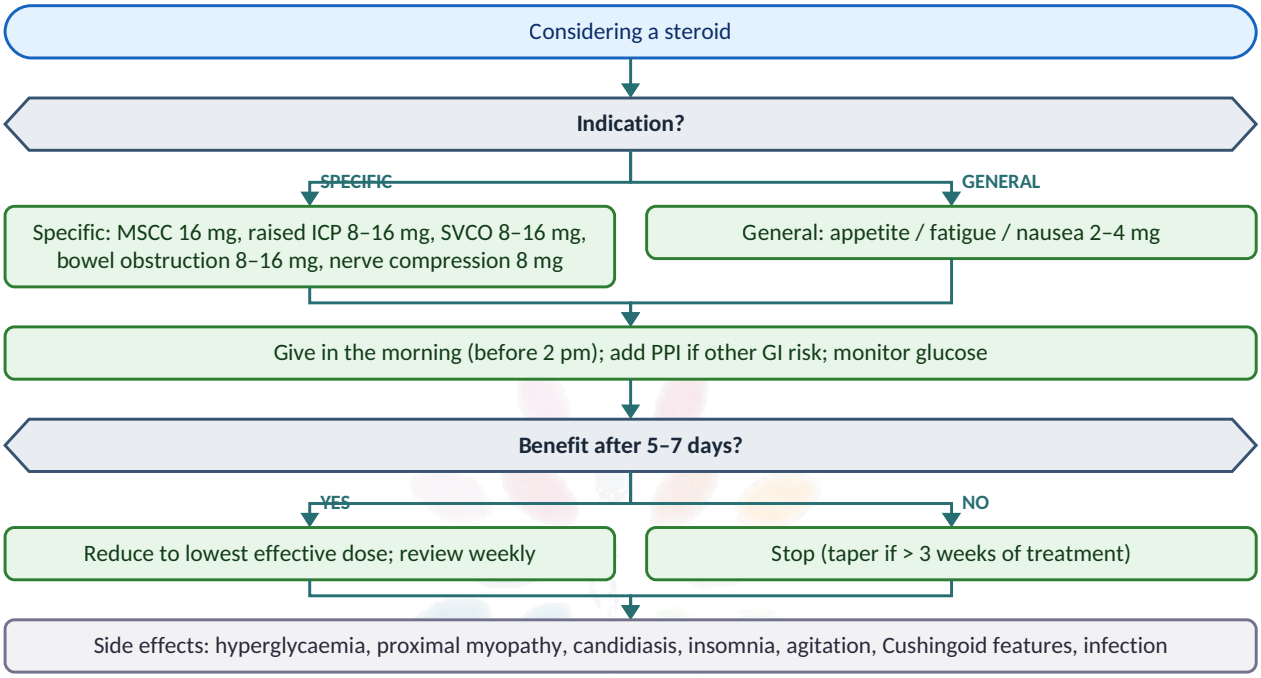
- NCCN Guidelines: Neuroendocrine and Adrenal Tumors (current version)

EXAM-ORIENTED THEORY

Palliative care focus — adrenocortical carcinoma

Problem	Management
Cushing syndrome (cortisol excess)	Metyrapone, ketoconazole, mitotane
Mitotane adrenal insufficiency	Hydrocortisone replacement at high doses
Hypokalaemia, hypertension, diabetes	Spironolactone, K replacement, insulin
Liver / lung metastases	Symptom control; prognosis often months

ALGORITHM · Use of Corticosteroids in Palliative Care Algo 60 — full page ›



★ KEY POINTS

- ▶ Proximal myopathy appears after 2–4 weeks and limits mobility.
- ▶ Steroid-induced hyperglycaemia peaks in the afternoon — check evening glucose.

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M92 Phaeochromocytoma / Paraganglioma (Malignant)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why must alpha-blockade precede beta-blockade in phaeochromocytoma management? [Paper 1, recurring]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply correct perioperative pharmacological sequencing to prevent hypertensive crisis (CD2.1, PD3)

DEFINITION Catecholamine-secreting tumours of the adrenal medulla (phaeochromocytoma) or extra-adrenal sympathetic/parasympathetic paraganglia (paraganglioma); malignancy is defined by the presence of metastasis (since histology alone cannot reliably predict behaviour), and a substantial proportion are linked to hereditary syndromes (SDHB mutations particularly associated with malignant behaviour).

Stage / Setting	Principal Treatment Approach
Localised, resectable	Surgical resection after mandatory pre-operative alpha-blockade (and beta-blockade if needed) to prevent catastrophic intraoperative hypertensive crisis
Metastatic, slow-growing	Observation or symptom-directed alpha/beta-blockade if indolent and minimally symptomatic
Metastatic, progressive/ symptomatic	Peptide receptor radionuclide therapy (if somatostatin/MIBG-avid), or combination chemotherapy (cyclophosphamide-vincristine-dacarbazine, CVD)

COMPLICATIONS

Disease-related:

- Hypertensive crisis, spontaneous or precipitated by anaesthesia/surgery/certain drugs, risking stroke, myocardial infarction or arrhythmia
- Catecholamine-induced cardiomyopathy from chronic excess catecholamine exposure
- Metastatic spread to bone, liver and lymph nodes (defining malignancy in this tumour type)
- Genetic syndrome-associated multi-organ tumours (MEN2, von Hippel-Lindau, SDHB-related paraganglioma syndromes) requiring broader screening

Treatment-related:

- Intraoperative hypertensive crisis if pre-operative alpha-blockade is inadequate or omitted – a critical, frequently tested perioperative safety point
- Severe hypotension after tumour removal as catecholamine levels suddenly fall, requiring careful perioperative fluid/vasopressor management
- Catecholamine surge during chemotherapy-induced tumour lysis, requiring continued alpha-blockade cover during treatment
- Cardiac complications from longstanding catecholamine-induced cardiomyopathy, sometimes only apparent after tumour removal

Drugs used — Drug · Indication · Dose · Caution

Phenoxybenzamine	Mandatory pre-operative alpha-blockade	10 mg orally twice daily, titrated upward over 1-2 weeks before surgery	Orthostatic hypotension, reflex tachycardia, nasal congestion
Cyclophosphamide + Vincristine + Dacarbazine (CVD)	Progressive metastatic disease	Per standard combination protocol	Myelosuppression, vincristine neuropathy, catecholamine release during treatment-induced tumour lysis

★ CLINICAL PEARLS & EXAM FOCUS

► Alpha-blockade must always precede beta-blockade in phaeochromocytoma management – giving a beta-blocker first can precipitate a hypertensive crisis from unopposed alpha-adrenergic effect.

► Genetic testing (SDHB and related genes) should be offered given the significant hereditary syndrome association and implications for family screening and malignancy risk assessment.

GUIDELINES & RECENT ADVANCES

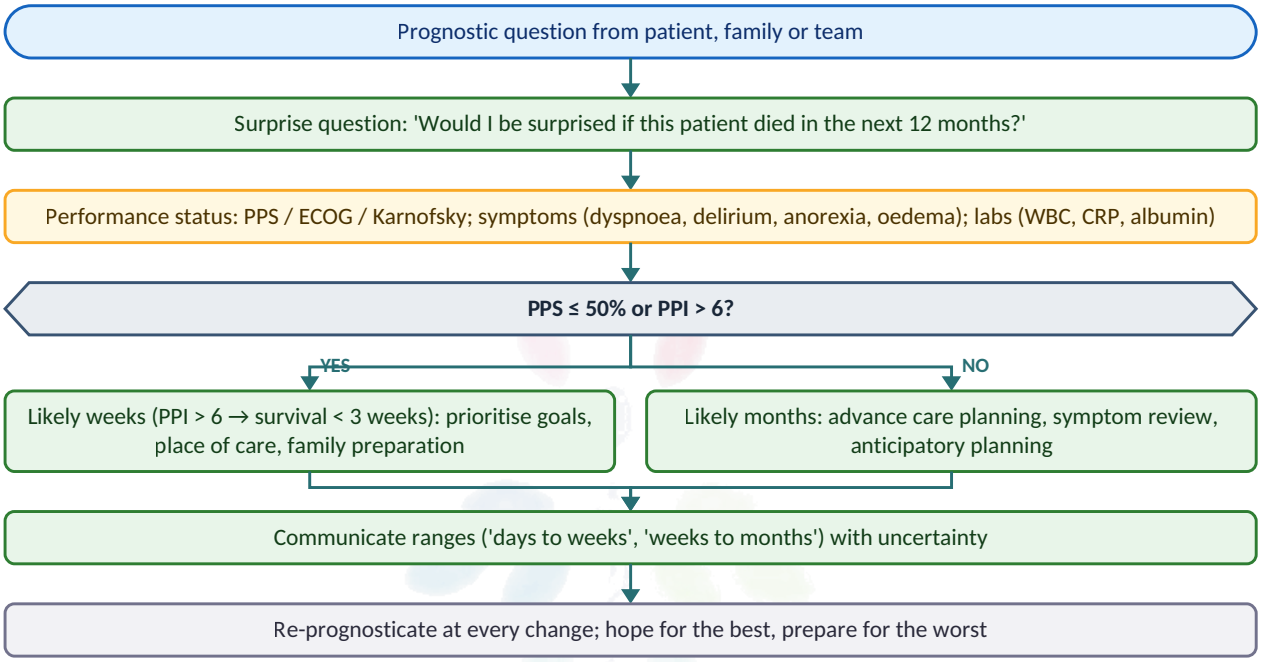
- Endocrine Society Clinical Practice Guideline: Pheochromocytoma and Paraganglioma

EXAM-ORIENTED THEORY

Palliative care focus — malignant phaeochromocytoma

Problem	Management
Catecholamine crises (hypertension, palpitations)	Alpha-blockade first (phenoxybenzamine / doxazosin), then beta-blocker
Drugs to avoid	Metoclopramide, glucagon, unopposed beta-blockers, some anaesthetic agents
Bone metastases	Radiotherapy, bone-targeted agents, MIBG therapy
Orthostatic hypotension on alpha-blockade	Liberal salt and fluid intake

ALGORITHM · Prognostication in Advanced Illness Algo 48 — full page ›



★ KEY POINTS

- ► PaP score uses dyspnoea, anorexia, KPS, CPS, WBC and lymphocyte %.
- ► Rate of change in function is a powerful prognostic clue.

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M93 Neuroblastoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What unique biological behaviour distinguishes infant neuroblastoma from most other malignancies? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise age- and biology-dependent risk stratification in paediatric oncology (CD8, CD2.1)

DEFINITION The commonest extracranial solid tumour of childhood, arising from primitive sympathetic neural crest cells (adrenal medulla or sympathetic chain), with a remarkably wide clinical spectrum from spontaneous regression in infants to highly aggressive, MYCN-amplified disease in older children.

Stage / Setting	Principal Treatment Approach
Low-risk disease (infants, favourable biology)	Surgery alone, or even observation in selected cases, given high rates of spontaneous regression/maturation
Intermediate-risk disease	Surgery combined with moderate-intensity chemotherapy
High-risk disease (MYCN-amplified, older child, metastatic)	Intensive multimodal therapy – induction chemotherapy, surgery, high-dose chemotherapy with autologous stem cell rescue, radiotherapy, and post-consolidation anti-GD2 immunotherapy with retinoic acid

COMPLICATIONS

Disease-related:

- Opsoclonus-myoclonus-ataxia syndrome, a distinctive paraneoplastic neurological complication (presumed autoimmune) seen even with low-stage disease
- Catecholamine secretion causing hypertension, sweating, and irritability
- Spinal cord compression from paraspinal tumour extension through intervertebral foramina ('dumbbell' tumours)
- Widespread metastasis (bone, bone marrow, liver, skin) in high-risk/stage 4 disease, sometimes with characteristic periorbital ecchymosis ('raccoon eyes') from orbital metastasis

Treatment-related:

- Severe pain during anti-GD2 immunotherapy infusions, requiring concurrent opioid analgesia as standard supportive care
- Capillary leak syndrome and hypotension during dinutuximab infusion, requiring close monitoring
- Growth and endocrine effects of intensive multimodal therapy (chemotherapy, radiotherapy, transplant) in young children
- Long-term hearing loss from platinum-based chemotherapy exposure

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dinutuximab (anti-GD2 antibody)		Per approved cyclical infusion	Severe infusion-related pain (neuropathic, given GD2 expression
	Post-consolidation immunotherapy, high-risk disease	protocol, combined with GM-CSF/IL-2 and isotretinoin	on peripheral nerves), capillary leak syndrome, hypersensitivity reactions
13-cis-Retinoic Acid (Isotretinoin)	Maintenance differentiation therapy, high-risk disease	160 mg/m²/day in divided doses, cyclical schedule	Dry skin/mucosa, hypertriglyceridaemia, teratogenicity

★ CLINICAL PEARLS & EXAM FOCUS

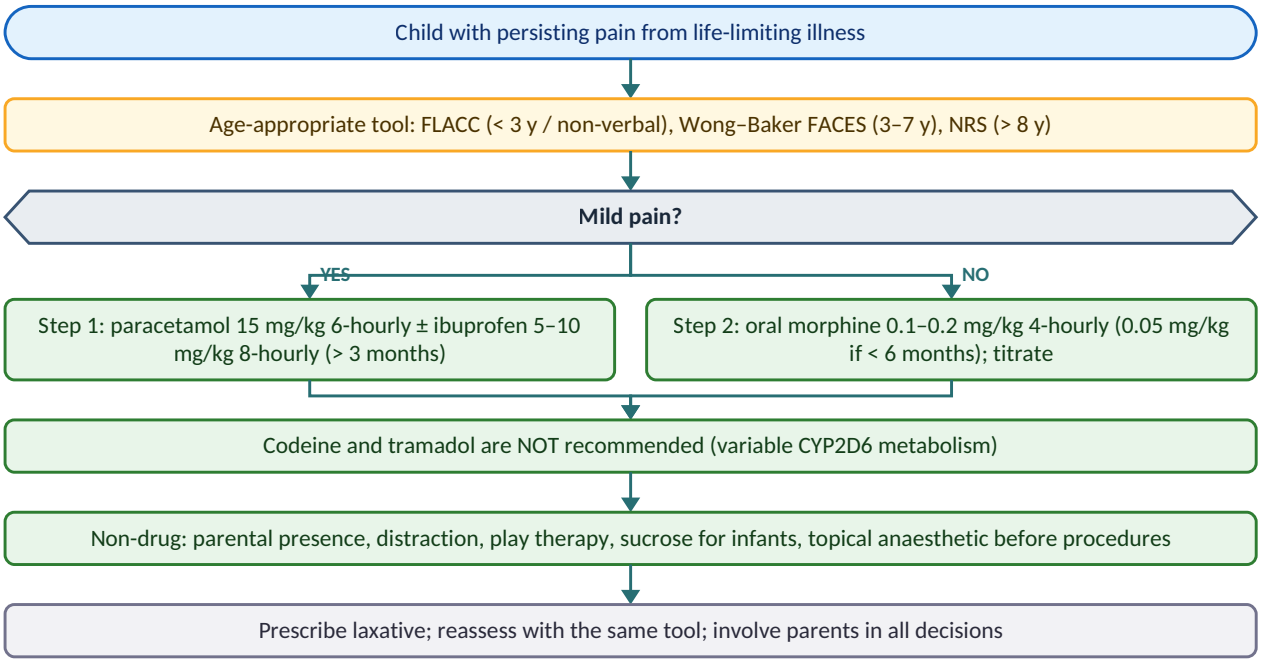
▸ Unlike almost any other malignancy, some infant neuroblastomas spontaneously regress or mature into benign ganglioneuroma without any treatment – a unique and frequently tested biological feature.

▸ Opsoclonus-myoclonus syndrome should prompt a search for an underlying (often small, low-stage) neuroblastoma even without other obvious tumour symptoms.

GUIDELINES & RECENT ADVANCES

- NCCN/COG Guidelines: Pediatric Neuroblastoma (current consensus)

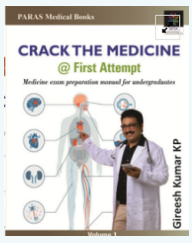
ALGORITHM · Pain Management in Children Algo 32 — full page ›



★ KEY POINTS

- ▸ Children are not small adults — dose by weight and age.
- ▸ ACT categories 1–4 classify life-limiting conditions for paediatric palliative care.

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M94 Wilms Tumour (Nephroblastoma)

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is gentle abdominal examination technique emphasised in suspected Wilms tumour? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply careful examination technique to avoid iatrogenic tumour rupture (CD8, PD1)

DEFINITION The commonest renal malignancy of childhood, arising from primitive metanephric blastema, typically presenting as an asymptomatic abdominal mass noticed by a caregiver, sometimes associated with congenital syndromes (WAGR, Beckwith-Wiedemann, Denys-Drash) and genitourinary anomalies.

Stage / Setting	Principal Treatment Approach
Localised, favourable histology	Nephrectomy followed by risk-adapted adjuvant chemotherapy (vincristine-actinomycin D, with or without doxorubicin based on stage)
Bilateral or unresectable at diagnosis	Pre-operative (neoadjuvant) chemotherapy to shrink the tumour, enabling later nephron-sparing or staged bilateral surgery
Anaplastic (unfavourable) histology or advanced stage	Intensified multi-agent chemotherapy with radiotherapy

COMPLICATIONS

Disease-related:

- Tumour rupture (spontaneous or intraoperative) causing peritoneal tumour spill and upstaging
- Renal vein/inferior vena cava tumour thrombus extension
- Hypertension from renin secretion by the tumour or renal compression
- Associated congenital anomalies (aniridia, genitourinary malformations, hemihypertrophy) that may need independent management

Treatment-related:

- Loss of renal function, particularly significant in bilateral disease requiring nephron-sparing surgery or bilateral nephrectomy with dialysis/transplant planning
- Cardiotoxicity from doxorubicin in higher-risk disease
- Radiation-related effects on growing tissues (musculoskeletal, gonadal) in children requiring radiotherapy
- Intraoperative tumour rupture during nephrectomy, upstaging the disease and intensifying subsequent treatment

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Vincristine + Actinomycin-D	Standard adjuvant chemotherapy backbone, favourable histology	Per standard risk-stratified protocol schedule	Vincristine neuropathy/ constipation, actinomycin-D mucositis/hepatotoxicity
Doxorubicin	Added for higher-stage/less favourable disease	Per protocol, cumulative dose monitored	Cardiotoxicity – requires baseline and serial echocardiography

★ CLINICAL PEARLS & EXAM FOCUS

▸ Careful, gentle abdominal palpation technique (avoiding vigorous examination) is taught specifically to avoid precipitating tumour rupture once Wilms tumour is suspected.

▸ Screening for associated congenital syndromes/anomalies should prompt consideration of contralateral kidney surveillance given bilateral disease risk in syndromic cases.

GUIDELINES & RECENT ADVANCES

- NCCN/COG Guidelines: Pediatric Renal Tumours (current consensus)

M95 Retinoblastoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why should radiotherapy be avoided where possible in hereditary retinoblastoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise germline cancer predisposition and its implications for treatment selection (CD8, CD9)

DEFINITION The commonest primary intraocular malignancy of childhood, arising from retinal precursor cells due to biallelic RB1 gene inactivation (hereditary bilateral or sporadic unilateral forms), classically presenting with leukocoria (a white pupillary reflex) noticed by parents, often in photographs.

Stage / Setting	Principal Treatment Approach
Small, intraocular tumour with useful vision potential	Eye-preserving (globe-salvage) focal therapy – laser photocoagulation, cryotherapy, or local chemotherapy (intra-arterial/intravitreal) combined with systemic chemotherapy
Large intraocular tumour, poor visual potential, or treatment failure	Enucleation (surgical removal of the eye) remains definitive and curative for disease confined to the globe
Extraocular extension/metastatic disease	Intensive systemic chemotherapy ± radiotherapy; prognosis significantly worse once disease extends beyond the globe

COMPLICATIONS

Disease-related:

- Vision loss, partial or complete, from the tumour itself or from treatment (enucleation)
- Extraocular extension along the optic nerve to the CNS, a major adverse prognostic development
- Trilateral retinoblastoma – a separate, synchronous pineal tumour in hereditary bilateral disease, requiring specific screening
- Second primary malignancies (particularly osteosarcoma and other sarcomas) in hereditary RB1 mutation carriers, especially within previously irradiated fields

Treatment-related:

- Loss of the affected eye (enucleation) with resultant cosmetic and functional impact, requiring prosthetic eye fitting and psychosocial support for the child and family
- Second malignancies as a major long-term concern specifically in hereditary (germline RB1 mutation) survivors, who should avoid radiotherapy where possible given markedly elevated second-cancer risk
- Ototoxicity from cumulative carboplatin exposure, requiring audiological monitoring
- Family screening implications given the hereditary form's autosomal dominant inheritance pattern

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Carboplatin + Etoposide + Vincristine	Systemic	Per standard risk-	
	chemotherapy for	stratified protocol	
	globe-salvage or adjuvant therapy		Myelosuppression, ototoxicity (carboplatin, cumulative), vincristine neuropathy
Melphalan (Intra-arterial)	Localised intra-arterial chemotherapy for globe salvage	Delivered via ophthalmic artery catheterisation per specialist protocol	Local vascular complications, myelosuppression, requires highly specialised interventional expertise

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★ CLINICAL PEARLS & EXAM FOCUS

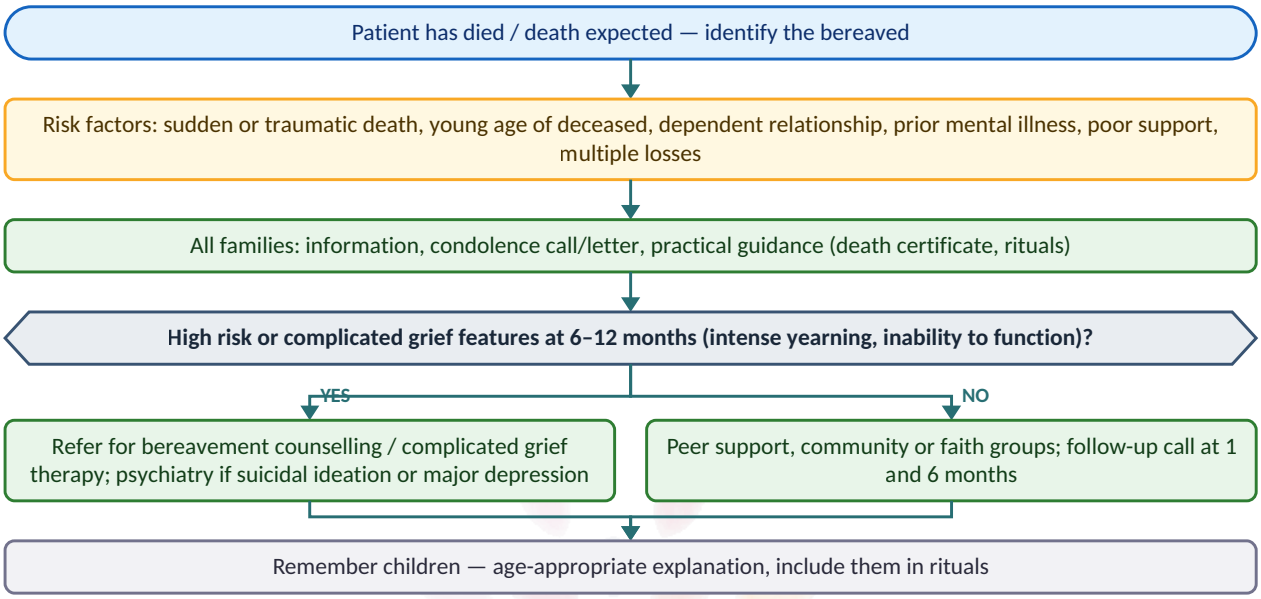
► Leukocoria noticed in a flash photograph is a classic, frequently examined presenting sign of retinoblastoma and should prompt urgent ophthalmological referral.

► Hereditary (germline RB1) retinoblastoma survivors carry a substantially elevated lifetime risk of second malignancies, particularly osteosarcoma, and radiotherapy should be avoided or minimised where alternative treatments exist.

GUIDELINES & RECENT ADVANCES

- NCCN/COG Guidelines: Pediatric Retinoblastoma (current consensus)

ALGORITHM · Bereavement Risk Assessment and Support Algo 38 — full page ›



★ KEY POINTS

► Prolonged grief disorder (ICD-11): persistent grief > 6 months with functional impairment.

► Kübler-Ross stages are not linear — dual process model is more useful.

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M96 Hepatoblastoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is alpha-fetoprotein particularly useful in hepatoblastoma? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply tumour marker-based monitoring in paediatric solid tumours (CD8, CD2.1)

DEFINITION The commonest primary liver malignancy of childhood, typically presenting before age 3 as an asymptomatic abdominal mass or distension, associated with prematurity, Beckwith-Wiedemann syndrome and familial adenomatous polyposis; alpha-fetoprotein is an excellent tumour marker for diagnosis and monitoring.

Stage / Setting	Principal Treatment Approach
Resectable at diagnosis	Surgical resection (partial hepatectomy) ± adjuvant chemotherapy depending on risk stratification
Unresectable at diagnosis	Neoadjuvant chemotherapy to shrink the tumour, enabling later resection or liver transplantation
Metastatic (commonly lung) or very high-risk disease	Intensive multi-agent chemotherapy combined with aggressive local control (resection/transplantation) of the primary

COMPLICATIONS

Disease-related:

- Pulmonary metastases, the dominant site of distant spread
- Tumour rupture causing intraperitoneal haemorrhage, a surgical emergency
- Vascular invasion (portal/hepatic vein) complicating resectability
- Paraneoplastic effects from beta-hCG secretion in rare variants, causing isosexual precocious puberty in boys

Treatment-related:

- Ototoxicity from cisplatin, a significant long-term concern given young patient age and impact on speech/language development
- Post-hepatectomy liver insufficiency, particularly relevant if extensive resection is required
- Growth and nutritional impact of intensive chemotherapy in infants/toddlers
- Transplant-related complications (rejection, immunosuppression-related infection) if liver transplantation is required for unresectable disease

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin + 5-Fluorouracil + Vincristine	Standard chemotherapy backbone (PLADO-type regimens also used)	Per standard risk-stratified paediatric protocol	Nephrotoxicity/ototoxicity (cisplatin), vincristine neuropathy, myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

► Alpha-fetoprotein is an outstanding tumour marker in hepatoblastoma – used for both diagnosis and monitoring treatment response/relapse, among the most reliable markers in all of paediatric oncology.

► Liver transplantation is a valid and often successful option for otherwise unresectable hepatoblastoma confined to the liver, distinguishing it favourably from many other childhood solid tumours.

GUIDELINES & RECENT ADVANCES

- NCCN/COG Guidelines: Pediatric Liver Tumours (current consensus)

M97 Langerhans Cell Histiocytosis

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What complication of Langerhans cell histiocytosis causes permanent endocrine deficit? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Recognise endocrine complications of hypothalamic-pituitary infiltrative disease (CD8, CD9.1)

DEFINITION A clonal proliferation of Langerhans-type dendritic cells, now classified as a neoplastic (myeloid-lineage) disorder, with a spectrum from a solitary bone lesion in older children to multisystem, life-threatening disease in infants; BRAF V600E mutation is found in a substantial proportion of cases.

Stage / Setting	Principal Treatment Approach
Single-system, single-site disease (e.g. solitary bone lesion)	Curettage ± intralesional corticosteroid injection, or observation alone, given frequent spontaneous resolution
Single-system, multifocal, or multisystem disease without risk-organ involvement	Systemic chemotherapy (vinblastine-prednisolone backbone) for a defined duration
Multisystem disease with risk-organ involvement (liver, spleen, bone marrow)	Intensive chemotherapy; BRAF inhibitor (vemurafenib) for refractory, BRAF-mutated disease

COMPLICATIONS

Disease-related:

- Diabetes insipidus from hypothalamic-pituitary infiltration, a common and sometimes permanent complication
- Lytic bone lesions causing pain and occasionally pathological fracture
- Risk-organ dysfunction (liver, spleen, haematopoietic) in multisystem disease, the key determinant of mortality risk
- Neurodegenerative LCH – a rare, delayed, progressive cerebellar/CNS complication occurring sometimes years after apparent disease resolution

Treatment-related:

- Permanent diabetes insipidus requiring lifelong desmopressin replacement, a common long-term sequela even after systemic disease is successfully treated
- Growth hormone deficiency and other hypopituitarism from hypothalamic-pituitary involvement
- Chemotherapy-related myelosuppression and infection risk during treatment
- Long-term neurological sequelae (neurodegenerative LCH) requiring surveillance even after apparent cure

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Vinblastine + Prednisolone	Standard first-line systemic chemotherapy	Per standard LCH-III/IV type protocol, weekly	Vincristine-class neuropathy (less with vinblastine), myelosuppression, steroid-related metabolic effects
		vinblastine with pulsed steroid	
Vemurafenib	Refractory, BRAF V600E-mutated multisystem disease	Weight-based paediatric dosing per protocol	Photosensitivity, rash, arthralgia, secondary cutaneous squamous lesions

★ CLINICAL PEARLS & EXAM FOCUS

► Diabetes insipidus in a child should prompt consideration of Langerhans cell histiocytosis, particularly if accompanied by lytic bone lesions or a skin rash.

► Risk-organ involvement (not the number of bone lesions) is the critical determinant of prognosis and treatment intensity in this disease.

GUIDELINES & RECENT ADVANCES

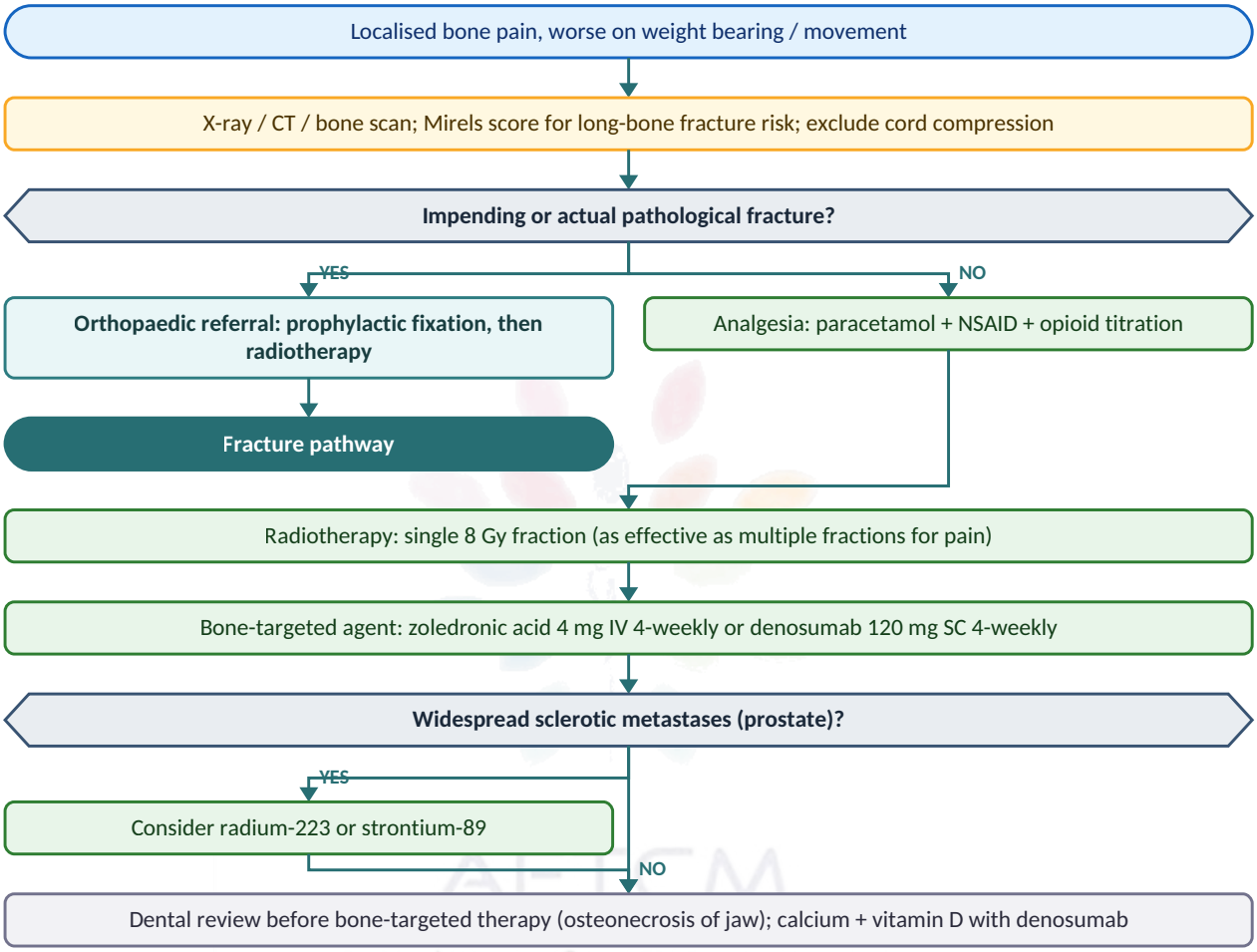
- Histiocyte Society Treatment Guidelines for Langerhans Cell Histiocytosis

EXAM-ORIENTED THEORY

Palliative care focus — Langerhans cell histiocytosis

Problem	Management
Lytic bone lesions with pain	NSAIDs, curettage, low-dose RT
Diabetes insipidus	Desmopressin
Skin lesions / seborrhoeic rash	Topical steroids
Pulmonary LCH (smokers)	Smoking cessation is key

ALGORITHM · Pain from Bone Metastases Algo 26 — full page ›



★ KEY POINTS

- ▶ Pain relief after radiotherapy takes 2–4 weeks; pain flare in the first days — cover with dexamethasone.
- ▶ Mirels score ≥ 9 → prophylactic fixation.

M98 Sinonasal and Paranasal Sinus Carcinoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What occupational exposures are linked to sinonasal malignancy? [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Link occupational history to specific malignancy risk in head and neck oncology (CD9, CD4.1)

DEFINITION A rare group of malignancies (squamous cell carcinoma, adenocarcinoma, olfactory neuroblastoma, and others) arising in the nasal cavity or paranasal sinuses, associated with occupational exposures (wood dust, nickel, leather), presenting late with non-specific sinonasal symptoms often mistaken for chronic sinusitis.

Stage / Setting	Principal Treatment Approach
Localised, resectable	Surgical resection (endoscopic or open craniofacial approach depending on extent) followed by adjuvant radiotherapy for high-risk features
Locally advanced, involving skull base/orbit	Multimodality therapy – surgery, radiotherapy and chemotherapy, often requiring combined neurosurgical-ENT approaches
Metastatic/unresectable	Platinum-based palliative chemotherapy; limited evidence base given rarity

COMPLICATIONS

Disease-related:

- Orbital invasion causing proptosis, diplopia or visual loss
- Skull-base erosion with intracranial extension and potential CSF leak/meningitis risk
- Cranial nerve deficits from local invasion along skull-base foramina
- Diagnostic delay given symptom overlap with benign chronic sinusitis, often resulting in advanced-stage presentation

Treatment-related:

- CSF leak and meningitis risk after skull-base surgical resection
- Visual and ocular complications after orbital-involving surgery or radiotherapy
- Radiation-induced damage to nearby critical structures (optic nerve/chiasm, brain, pituitary) given close anatomical proximity
- Facial disfigurement after extensive craniofacial resection, with significant psychosocial impact

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Cisplatin	Concurrent chemoradiation or palliative chemotherapy	100 mg/m² every 3 weeks (concurrent) or per palliative schedule	Nephrotoxicity, ototoxicity, myelosuppression

★ CLINICAL PEARLS & EXAM FOCUS

► Persistent unilateral nasal obstruction, bleeding, or facial pain/numbness that fails to resolve with standard sinusitis treatment warrants imaging and ENT referral to exclude malignancy.

► A relevant occupational history (wood dust, nickel, leather tanning) should always be sought given well-established associations with specific sinonasal cancer subtypes.

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Head and Neck Cancers (Nasal Cavity and Paranasal Sinus section, current version)

M99 Cancer of Unknown Primary

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Why is identifying a 'favourable subset' of cancer of unknown primary clinically important? [Paper 4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Apply structured diagnostic algorithms to guide treatment in occult primary malignancy (CD2.1, CD4.1)

DEFINITION A clinical scenario where metastatic carcinoma is confirmed histologically but the primary site cannot be identified despite thorough clinical, radiological and pathological evaluation, accounting for a meaningful proportion of all cancer presentations and requiring a structured diagnostic and treatment approach.

Stage / Setting	Principal Treatment Approach
Favourable subset (e.g. women with axillary nodal adenocarcinoma, peritoneal papillary serous carcinomatosis, men with blastic bone metastases and elevated PSA)	Site-specific treatment as if the presumed primary (breast, ovarian, prostate respectively) were confirmed, given recognised favourable response patterns
Unfavourable, poorly differentiated carcinoma	Empirical platinum-based combination chemotherapy, extrapolated from regimens effective across multiple primary sites
Unfavourable, well-differentiated adenocarcinoma, no favourable features identified	Empirical chemotherapy (platinum-taxane based) with ongoing search for an emergent primary site, and molecular profiling to guide therapy where available

COMPLICATIONS

Disease-related:

- Rapid, often multi-site metastatic progression given typically aggressive tumour biology in this group
- Site-specific complications depending on dominant metastatic burden (bone pain/fracture, malignant effusions, hepatic failure, brain metastases)
- Significant psychological distress from the diagnostic uncertainty itself ('not knowing' the origin of one's cancer)
- Delayed identification of a treatable primary (e.g. lymphoma, germ cell tumour) if adequate immunohistochemical/molecular workup is not pursued

Treatment-related:

- Standard platinum-taxane chemotherapy toxicities (myelosuppression, neuropathy)
- Missed treatable favourable subtypes (germ cell tumour, lymphoma) if comprehensive immunohistochemistry/molecular testing is skipped in the initial workup – a critical diagnostic pitfall
- Psychosocial burden from prognostic uncertainty complicating communication and goals-of-care discussions
- Treatment-related toxicity in patients whose overall prognosis may be limited, requiring careful risk-benefit discussion

Drugs used — Drug · Indication · Dose · Caution

Carboplatin + Paclitaxel	Empirical first-line chemotherapy, unfavourable subset	Carboplatin AUC 5 + paclitaxel 175-200 mg/m² every 3 weeks	Myelosuppression, peripheral neuropathy

★ CLINICAL PEARLS & EXAM FOCUS

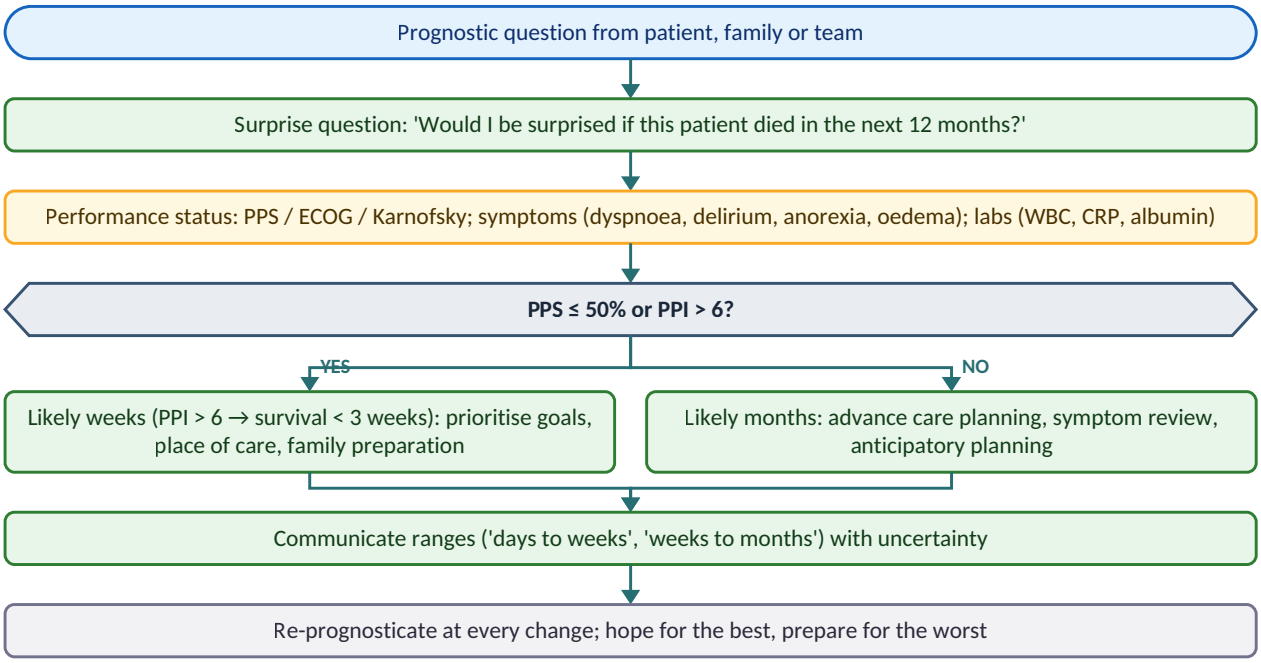
► A thorough search for 'favourable subset' features (specific marker patterns, nodal distribution) is essential, since recognising these can change treatment from empirical palliative chemotherapy to potentially curative, site-specific therapy.

► Comprehensive immunohistochemistry and, where available, molecular/gene-expression profiling should always be pursued before labelling a cancer definitively 'unknown primary', to avoid missing a highly treatable tumour type (germ cell, lymphoma).

GUIDELINES & RECENT ADVANCES

- NCCN Guidelines: Occult Primary (current version)

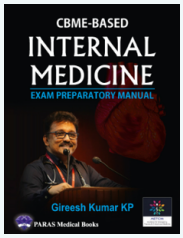
ALGORITHM · Prognostication in Advanced Illness Algo 48 — full page ›



★ KEY POINTS

- ▶ PaP score uses dyspnoea, anorexia, KPS, CPS, WBC and lymphocyte %.
- ▶ Rate of change in function is a powerful prognostic clue.

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M100 Choroidal (Uveal) Melanoma

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How does the biology and metastatic pattern of uveal melanoma differ from cutaneous melanoma? [Paper 4, recent advances]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Distinguish uveal from cutaneous melanoma biology to guide surveillance and treatment choice (CD2.1, CD4.1)

DEFINITION The commonest primary intraocular malignancy in adults, arising from melanocytes within the choroid, ciliary body, or iris, typically detected on routine fundoscopic examination or presenting with blurred vision/ visual field defect; biologically and genetically distinct from cutaneous melanoma, with a strong propensity for haematogenous (particularly hepatic) rather than lymphatic spread.

Stage / Setting	Principal Treatment Approach
Small, localised tumour with vision-preserving potential	Plaque brachytherapy (episcleral radioactive plaque) as the globe- and vision-preserving standard for most small-to-medium tumours
Large tumour or treatment failure	Enucleation (surgical removal of the eye), reserved for tumours too large for radiotherapy or with painful blind eye/extraocular extension
Metastatic (liver-dominant) disease	Liver-directed therapy (hepatic artery embolisation/infusion) for liver-confined disease; immunotherapy (including tebentafusp for HLA-A*02:01-positive eligible patients) for systemic disease, given generally poor response to melanoma regimens effective in cutaneous disease

COMPLICATIONS

Disease-related:

- Liver metastases, occurring in the majority of patients who develop metastatic disease, reflecting the eye's venous drainage pattern bypassing lymphatics
- Retinal detachment from tumour growth, causing progressive visual field loss
- Secondary glaucoma from tumour-related angle closure or vitreous haemorrhage
- Radiation retinopathy/optic neuropathy as a late complication of plaque brachytherapy, causing delayed vision loss even after successful tumour control

Treatment-related:

- Cytokine release syndrome during initial tebentafusp infusions, requiring monitored inpatient administration for early doses
- Radiation-induced retinopathy and cataract after plaque brachytherapy, sometimes outweighing initial vision-preservation benefit over time
- Vision loss from enucleation, with psychosocial and functional impact requiring prosthetic eye fitting and adaptation support
- Poor response of metastatic uveal melanoma to standard cutaneous melanoma immunotherapy regimens, historically a major therapeutic challenge now partly addressed by tebentafusp

Trauma & Critical care Medicine

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Tebentafusp	Metastatic disease, HLA-A*02:01-positive patients	Per approved weight-based weekly IV infusion schedule	Cytokine release syndrome (requires inpatient monitoring for initial doses), skin rash, hypotension

★ CLINICAL PEARLS & EXAM FOCUS

► Uveal melanoma is biologically distinct from cutaneous melanoma (different driver mutations, GNAQ/ GNA11 rather than BRAF, and near-exclusive haematogenous spread to liver) – standard cutaneous melanoma checkpoint inhibitor regimens are generally far less effective here, an important distinction for exams.

► Regular liver surveillance (imaging/liver function tests) is recommended in uveal melanoma survivors given the strong, specific predilection for late hepatic metastasis.

GUIDELINES & RECENT ADVANCES

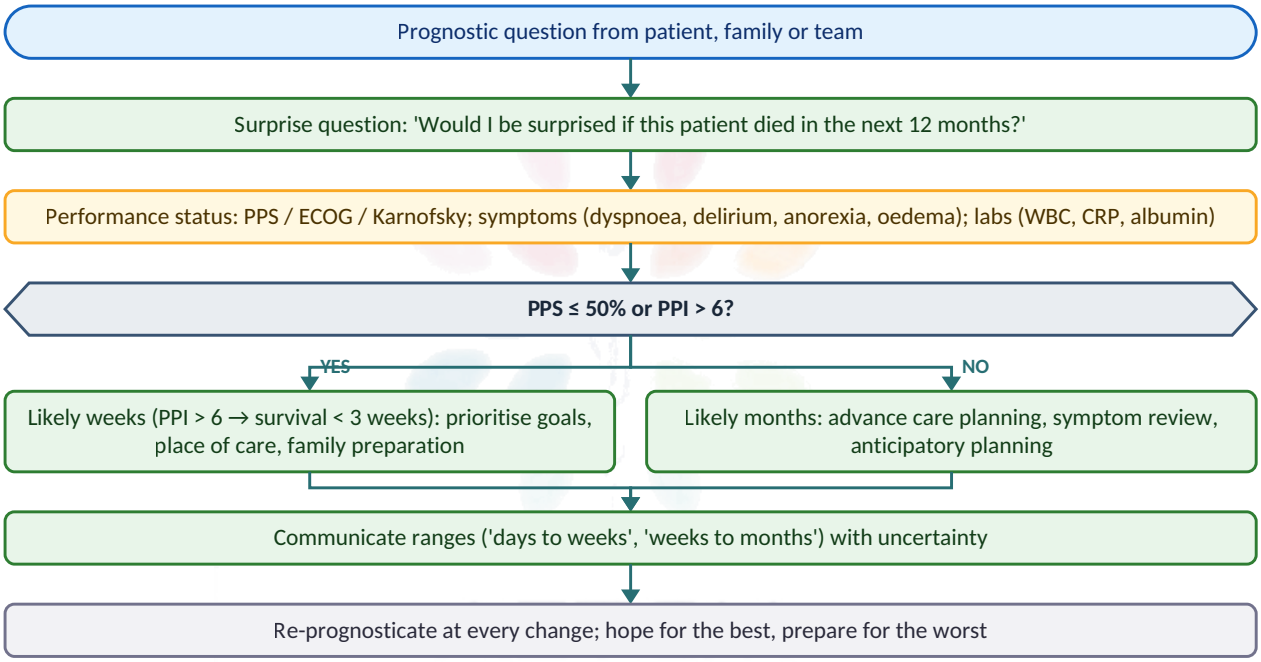
- NCCN Guidelines: Uveal Melanoma (current version)

EXAM-ORIENTED THEORY

Palliative care focus — uveal melanoma

Problem	Management
Liver metastases (> 90% of metastatic cases)	Liver-directed therapy, tebentafusp (HLA-A*02:01), symptom care
Painful blind eye	Enucleation for comfort; topical steroids/atropine
Hepatic capsular pain	Dexamethasone, opioids
Monocular adjustment	Low-vision rehabilitation

ALGORITHM · Prognostication in Advanced Illness Algo 48 — full page ›



★ KEY POINTS

► PaP score uses dyspnoea, anorexia, KPS, CPS, WBC and lymphocyte %.

► Rate of change in function is a powerful prognostic clue.

SECTION 3

Pain Management in Palliative Medicine

Pain management spans pathophysiology, comprehensive clinical assessment, advanced pharmacotherapy, interventional techniques and holistic psychosocial care. Kept here as a dedicated section — separate from the general CBME curriculum (Part I) and the Question & Answer practice (Part III) — with one full page devoted to each of its nineteen sub-topics.

3.1 Pathophysiology & Classification of Pain 235

3.2 Clinical Assessment & Measurement 241

3.3 Pharmacological Management 246

3.4 Interventional & Disease-Modifying Therapies 256

3.5 Pain Emergencies & Complex Scenarios 261

3.6 Non-Pharmacological & Rehabilitative Approaches 265

AT A GLANCE

Pain management at a glance

Principle	Practice
By the mouth	Oral route preferred; SC when oral fails
By the clock	Regular dosing for continuous pain + PRN rescue
By the ladder	Match drug to severity; low-dose strong opioid may replace step 2
For the individual	Titrate to effect and tolerability
Attention to detail	Laxative with every opioid; review in 24–72 h
Total pain	Physical, psychological, social and spiritual domains

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SECTION 3 · CHAPTER 3.1

Pathophysiology & Classification of Pain

The neurobiological and conceptual foundation for every pain-management answer in the examination.

3.1.1 Neurobiology of Pain 236

3.1.2 Pain Syndromes & Types 238

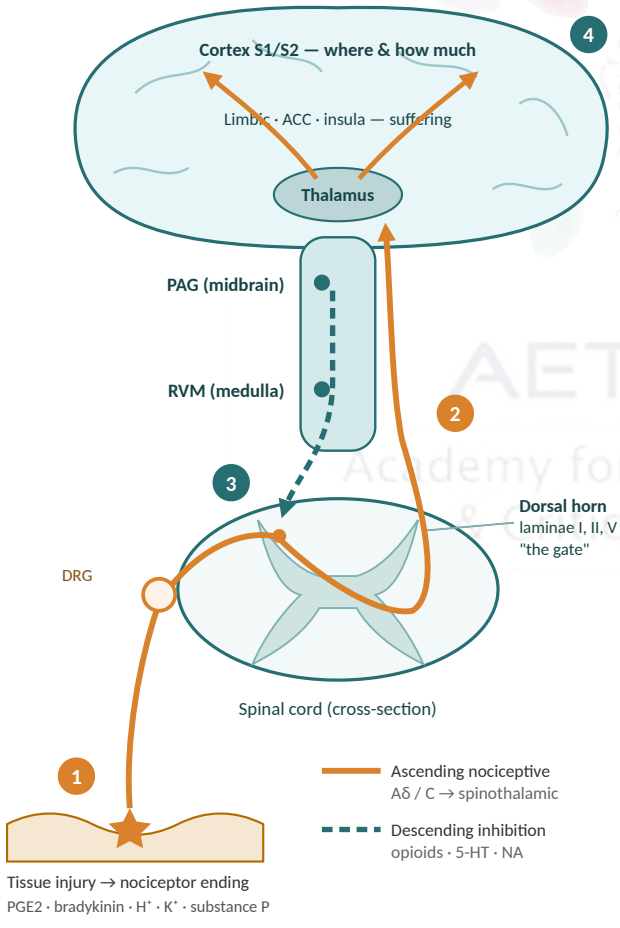
3.1.3 Concept of Total Pain 239

AT A GLANCE

Pain types at a glance

Type	Features	Responds to
Somatic nociceptive	Well-localised, aching (bone, soft tissue)	Paracetamol, NSAID, opioid, RT
Visceral nociceptive	Poorly localised, referred, colicky	Opioid, antispasmodic, plexus block
Neuropathic	Burning, shooting, allodynia, numbness	TCA, gabapentinoid, SNRI, steroid
Mixed	Features of both	Opioid + adjuvant
Nociplastic	Altered nociception without clear damage	Multimodal, non-drug therapies

FIGURE — THE PAIN PATHWAY (TTMP)



1 TRANSDUCTION
Noxious stimulus becomes an electrical signal at free nerve endings (TRPV1, ASIC, $Na_V1.7$). Sensitised by PGE2, bradykinin, histamine, H^+, K^+.
Act here: NSAIDs, steroids, topical LA, capsaicin.

2 TRANSMISSION
Aδ fibres (myelinated, fast, sharp "first pain") and C fibres (unmyelinated, slow, dull "second pain") → DRG → dorsal horn → cross the midline → spinothalamic tract → thalamus.
Act here: local anaesthetics, nerve blocks, neurolysis.

3 MODULATION
Dorsal-horn "gate" (Melzack & Wall, 1965). Descending inhibition PAG → RVM using endogenous opioids, serotonin, noradrenaline. NMDA-driven wind-up causes central sensitisation.
Act here: opioids, TCAs/SNRIs, gabapentinoids, ketamine.

4 PERCEPTION
Thalamus → S1/S2 (location, intensity) plus limbic system, ACC, insula (emotion, suffering) — where "total pain" acts.
Act here: explanation, psychological & spiritual care, CBT.

3.1.1 Neurobiology of Pain

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** Describe the pain pathway. Discuss transduction, transmission, modulation and perception. [Paper 1 — Basic Sciences]
- **Q** Write short notes on peripheral and central sensitisation and their clinical relevance to chronic pain. [Paper 1, recurring theme]

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe the neuroanatomy and neurophysiology of nociception (CD3.1.2, CD3.1.3)
- ✓ Relate pain to underlying pathophysiology to plan rational pharmacological and non-pharmacological treatment (PD3.3)

DEFINITION Nociception is the neural process of encoding and processing noxious stimuli. Pain is "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" (IASP, 2020) – always a subjective, biopsychosocial experience, not merely a nociceptive signal.

Step	Location	Mechanism
Transduction	Peripheral nociceptor terminals (Aδ and C fibres)	Noxious mechanical/thermal/chemical stimuli converted
		to an electrical signal via TRPV1, ASIC, P2X and other
		transducer channels
Transmission	Peripheral nerve → dorsal root ganglion → dorsal	Action potentials conducted centrally; first synapse in
	horn → spinothalamic tract → thalamus → cortex	dorsal horn laminae I, II and V
Modulation	Dorsal horn (gate-control) and descending	Facilitation or inhibition of nociceptive transmission via
	pathways (periaqueductal grey, rostral	endogenous opioids, serotonin, noradrenaline
	ventromedial medulla)	(descending inhibition)
Perception	Somatosensory cortex, limbic system, prefrontal	Integration of sensory, affective and cognitive
	cortex	components into the conscious experience of pain

FEATURE	PERIPHERAL SENSITISATION	CENTRAL SENSITISATION
Site	Nociceptor terminal	Dorsal horn neurons / CNS
Mechanism	Inflammatory mediators (prostaglandins, bradykinin, histamine, cytokines) lower nociceptor threshold	NMDA-receptor-mediated 'wind-up'; increased synaptic efficacy and receptive field expansion
Clinical correlate	Primary hyperalgesia (tenderness at the site of injury)	Secondary hyperalgesia and allodynia (pain from a non-painful stimulus) spreading beyond the injury site

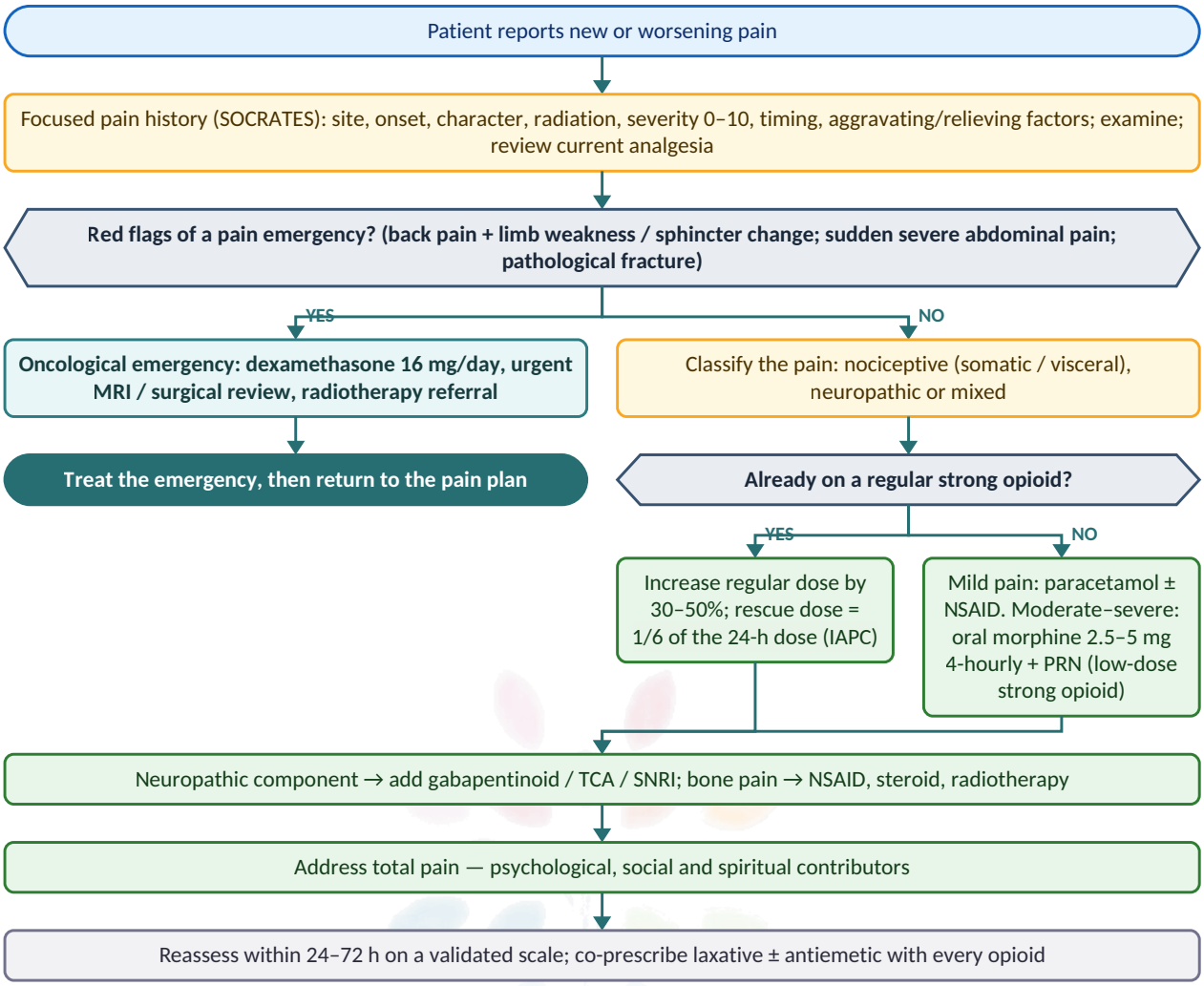
★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Mnemonic for the pain pathway: 'TTMP' – Transduction, Transmission, Modulation, Perception.
- ▸ NMDA-receptor antagonists (e.g. ketamine) specifically target central sensitisation/wind-up – the pharmacological rationale for their use in refractory neuropathic pain.

GUIDELINES & RECENT ADVANCES

- IASP Terminology (revised definition of pain, 2020)

ALGORITHM · Approach to New or Worsening Pain in a Palliative Care Patient Algo 1 — full page ›



★ KEY POINTS

- ▸ Always exclude a pain emergency first — spinal cord compression and pathological fracture are time-critical.
- ▸ Titrate by 30–50% steps; 'by the clock, by the mouth, by the ladder, for the individual'.

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3.1.2 Pain Syndromes & Types

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Classify pain. Differentiate nociceptive from neuropathic pain with examples. [Paper 2]

Q Enumerate cancer-related pain syndromes. Describe chemotherapy-induced peripheral neuropathy. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Differentiate somatic, visceral, neuropathic, bone and mixed pain (CD3.1.1, CD3.1.6, CD3.1.8)

✓ Recognise specific cancer pain syndromes such as plexopathies, mucositis and CIPN (CD3.1.5)

DEFINITION Pain is classified by underlying mechanism (nociceptive vs neuropathic vs nociplastic), by duration (acute vs chronic), and by cancer relationship (tumour-related, treatment-related or incidental/unrelated).

Type	Mechanism	Character	Example
Somatic	Nociceptor activation in skin, muscle, bone	Well-localised, aching, throbbing	Bone metastasis, post-surgical wound pain
Visceral	Nociceptor activation in a hollow/solid viscus	Poorly localised, deep, cramping, referred	Malignant bowel obstruction, hepatic capsular pain
Neuropathic	Damage/dysfunction of the somatosensory nervous system	Burning, shooting, electric-shock-like; allodynia, hyperalgesia	CIPN, post-herpetic neuralgia, plexopathy
Bone	Combination of nociceptive, inflammatory and neuropathic mechanisms	Constant background ache with incident/movement-related flares	Vertebral or long-bone metastasis
Mixed	Simultaneous nociceptive + neuropathic mechanisms	Combination of above features	Malignant plexopathy invading soft tissue and nerve

Key cancer-related pain syndromes

- **Plexopathies** – brachial (Pancoast tumour, breast cancer nodal disease) and lumbosacral (pelvic/ retroperitoneal tumours) plexus invasion.
- **Oral mucositis** – chemo-/radiotherapy-induced epithelial injury; severe nociceptive pain limiting oral intake.
- **Chemotherapy-induced peripheral neuropathy (CIPN)** – length-dependent, symmetric, glove-and-stocking distribution; classically with platinum agents, taxanes and vinca alkaloids.

★ CLINICAL PEARLS & EXAM FOCUS

► 'Red-flag' pain (sudden-onset severe back pain + leg weakness, or acute dyspnea + chest pain) should always trigger evaluation for an oncological pain emergency (Section 4, Algorithm Atlas).

GUIDELINES & RECENT ADVANCES

- NCCN Clinical Practice Guidelines: Adult Cancer Pain

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3.1.3 Concept of Total Pain

Practice Qs ›
Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Describe the concept of 'Total Pain' as proposed by Dame Cicely Saunders. [Paper 1, frequently asked]
- Q How do physical, psychological, social and spiritual distress interact to amplify the perception of pain? [Paper 1/3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Explain the role of psychological, social and spiritual issues in pain and suffering (Subject-specific Objective 2)
- ✓ Evaluate how physical, psychological, social and spiritual distress interact to amplify pain (CD3.1.4)

DEFINITION "Total Pain" (Dame Cicely Saunders, St Christopher's Hospice, 1960s) describes the composite suffering of a patient with advanced illness, in which physical nociception is inseparably compounded by psychological distress, social concerns and spiritual/existential suffering.

Domain	Typical Contributing Factors
Physical	Uncontrolled nociceptive/neuropathic pain, other symptom burden, treatment side-effects, fatigue
Psychological	Fear of dying, anxiety, depression, anger, loss of control, unresolved grief
Social	Role loss (breadwinner, parent), financial strain, isolation, family conflict, unfinished business
Spiritual/ Existential	Loss of meaning and hope, questions of why, guilt, disrupted relationship with the sacred/ transcendent

Why this matters in the examination

A 'total pain' assessment explains why a pain score can remain high despite an apparently adequate opioid dose, and justifies the mandatory multidisciplinary (physician, nurse, social worker, chaplain, psychologist) approach to refractory pain – a theme that recurs across Paper 1, 2 and 3 long-essay questions.

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Quote Saunders directly in an examination essay: "You matter because you are you, and you matter to the last moment of your life."
- ▸ A rising opioid requirement with no new organic cause should always prompt a total-pain / psychosocial reassessment before further dose escalation.

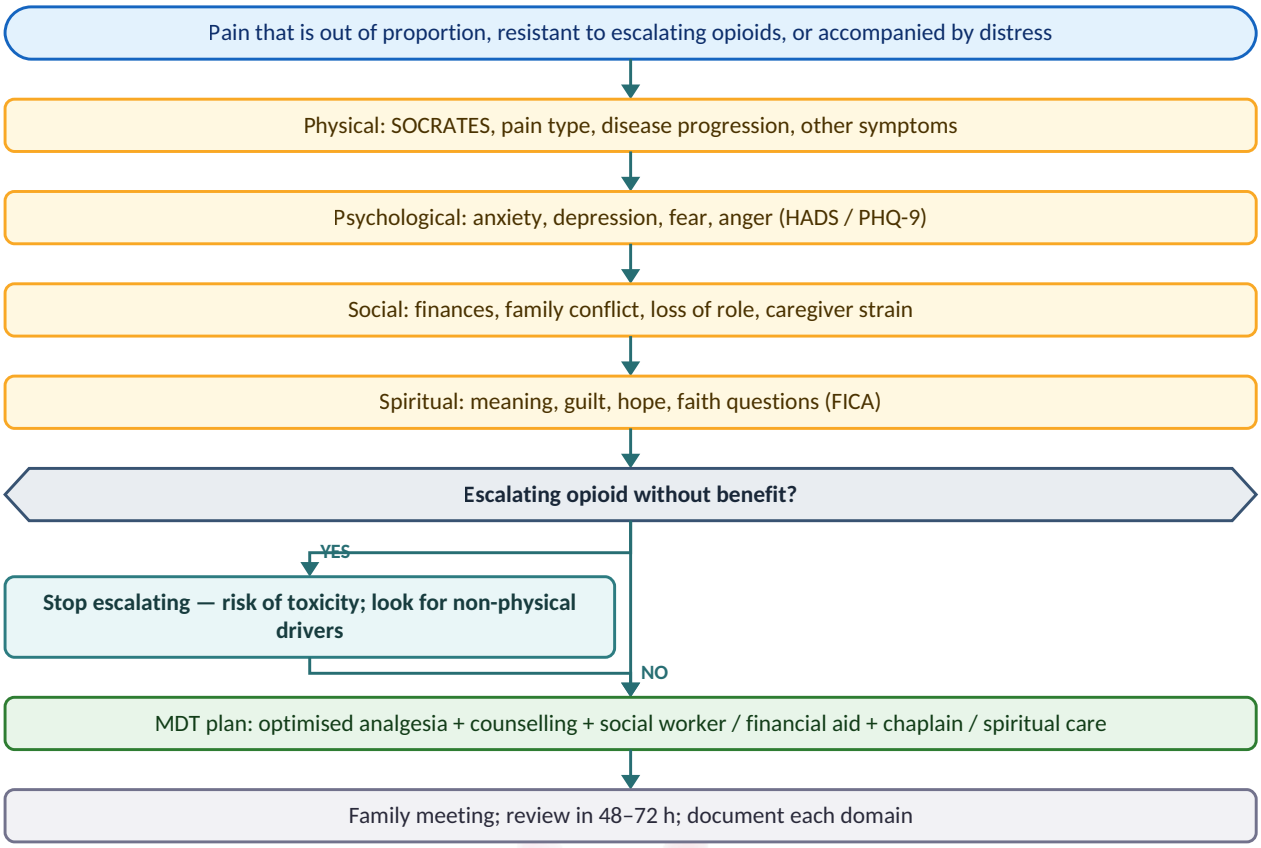
GUIDELINES & RECENT ADVANCES

- Saunders C. The evolution of palliative care. *J R Soc Med* 2001 (historical reference, freely indexed on PubMed)

FIGURE — TOTAL PAIN (DAME CICELY SAUNDERS)



ALGORITHM · Assessment and Management of Total Pain Algo 31 — full page ›



★ **KEY POINTS**

- Chemical coping (using opioids for emotional distress) signals unaddressed total pain.
- Ask: 'What worries you most?' — often reveals the hidden domain.

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SECTION 3 · CHAPTER 3.2

Clinical Assessment & Measurement

Structured history-taking, validated tools and special considerations for non-communicative, cognitively impaired and paediatric patients.

3.2.1 Multidimensional Assessment 242

3.2.2 Validated Assessment Tools 243

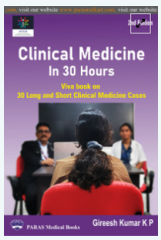
3.2.3 Special Populations 244

AT A GLANCE

Assessment tools at a glance

Tool	Use
NRS / VAS / VRS	Pain intensity (self-report)
Brief Pain Inventory	Intensity + interference
DN4 / LANSS	Neuropathic pain screening
ESAS-r	Multi-symptom screening
PAINAD / Abbey	Dementia, non-verbal adults
FLACC / Wong–Baker FACES	Children
Edmonton Classification System for Cancer Pain	Prognostic factors for pain control

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3.2.1 Multidimensional Assessment

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the clinical assessment of a patient presenting with pain in a palliative care setting. [Paper 2]

Q What is SOCRATES? Discuss its application to cancer pain assessment. [Viva, frequently asked]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Take a structured pain history and perform a targeted examination (PD3.1, PD3.2)

✓ Rationalise investigations in a patient with pain (PD3.4)

DEFINITION Multidimensional pain assessment is the structured evaluation of a pain complaint across its sensory (site, character, severity), temporal, and total-pain (psychosocial/spiritual) dimensions, supplemented by a focused neurological and musculoskeletal examination.

History – the SOCRATES framework

Letter	Domain	Example Question
S	Site	Where exactly is the pain?
O	Onset	When did it start? Sudden or gradual?
C	Character	Aching, burning, stabbing, cramping?
R	Radiation	Does it spread anywhere?
A	Associated features	Nausea, weakness, numbness, bowel/bladder symptoms?
T	Time course	Constant, intermittent, worse at a particular time?
E	Exacerbating/relieving factors	Movement, position, previous analgesics tried?
S	Severity	0–10 numerical rating at rest and on movement

Examination & targeted investigation

- Focused neurological examination for any suspected neuropathic component or cord compression (power, sensation, reflexes, sphincter tone).
- Musculoskeletal examination for localised bone tenderness, pathological fracture risk, joint effusion.
- Investigate *only* where the result will change management – e.g. plain film/MRI for suspected pathological fracture or cord compression, not routinely for every new pain complaint in a dying patient.

★ CLINICAL PEARLS & EXAM FOCUS

► Always document pain severity both at rest and on movement/incident activity – the two frequently require different management strategies (background opioid vs breakthrough dosing).

GUIDELINES & RECENT ADVANCES

- IAPC Standards for Palliative Care in India – assessment domain

Trauma & Critical care Medicine

3.2.2 Validated Assessment Tools

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Enumerate pain assessment tools used in palliative care. Describe the Brief Pain Inventory. [Paper 2]

Q Differentiate unidimensional from multidimensional pain assessment tools. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply validated unidimensional and multidimensional pain-assessment instruments (CD1.7.1)

DEFINITION A validated pain-assessment tool is a standardised, reproducible instrument used to quantify pain intensity (unidimensional) or its broader sensory, affective and functional impact (multidimensional).

Tool	Type	Principal Use
Numerical Rating Scale (NRS)	Unidimensional	0–10 self-report; the most widely used clinical and research scale
Visual Analogue Scale (VAS)	Unidimensional	100 mm line anchored 'no pain' to 'worst pain'; used mainly in research
Wong-Baker FACES Scale	Unidimensional (pictorial)	Children ≥3 years and adults with limited literacy/language
Brief Pain Inventory (BPI)	Multidimensional	Severity + interference with function, mood, sleep, relationships; gold standard in cancer-pain trials
McGill Pain Questionnaire	Multidimensional	Sensory/affective/evaluative word-descriptors; characterises neuropathic quality
Edmonton Symptom Assessment System (ESAS)	Multidimensional (multi-symptom)	9 symptoms including pain, scored 0–10; routine palliative-care vital-sign-style tool

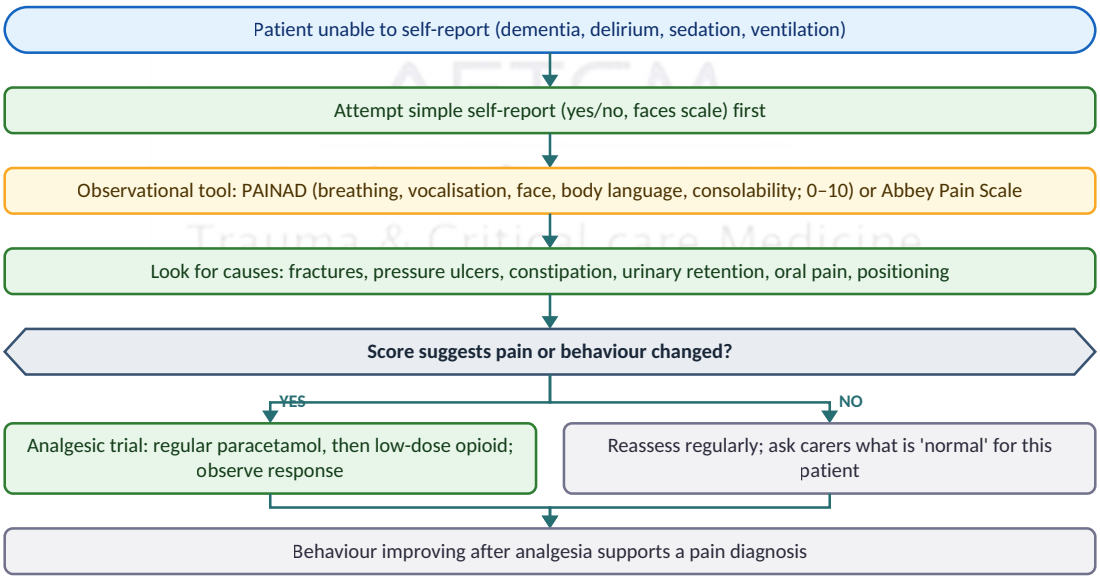
★ CLINICAL PEARLS & EXAM FOCUS

► The ESAS is the single most-quoted multi-symptom tool across the whole Palliative Medicine syllabus – learn it once, cite it everywhere (pain, dyspnea, nausea, fatigue, anxiety, depression, drowsiness, appetite, well-being).

GUIDELINES & RECENT ADVANCES

- Hui D, Bruera E. The Edmonton Symptom Assessment System 25 years later. *J Pain Symptom Manage* 2017

ALGORITHM · Pain Assessment in Non-Verbal and Cognitively Impaired Patients Algo 45 — full page ›



3.2.3 Special Populations

Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How do you assess pain in a non-communicative patient with advanced dementia? [Paper 3]

Q Describe pain assessment in the paediatric palliative care population. [Paper 2/3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Assess pain in non-communicative patients and those with advanced dementia (CD1.7 scales; CD8.1.2)

✓ Recognise developmental influences on pain assessment in children (AD8.3)

DEFINITION Special-population pain assessment uses observational, behavioural or proxy-reported tools when self-report is unreliable or impossible – in advanced dementia, delirium, profound disability, or in infants and young children.

Population	Tool	Domains Observed
Advanced dementia / non-verbal adult	PAINAD (Pain Assessment in Advanced Dementia)	Breathing, negative vocalisation, facial expression, body language, consolability (0–10)
Infants / pre-verbal children	FLACC scale	Face, Legs, Activity, Cry, Consolability
Children 3–7 years	Wong-Baker FACES / Faces Pain Scale-Revised	Pictorial self-report
Critically ill / sedated adult	Critical-Care Pain Observation Tool (CPOT)	Facial expression, body movement, muscle tension, ventilator compliance

★ CLINICAL PEARLS & EXAM FOCUS

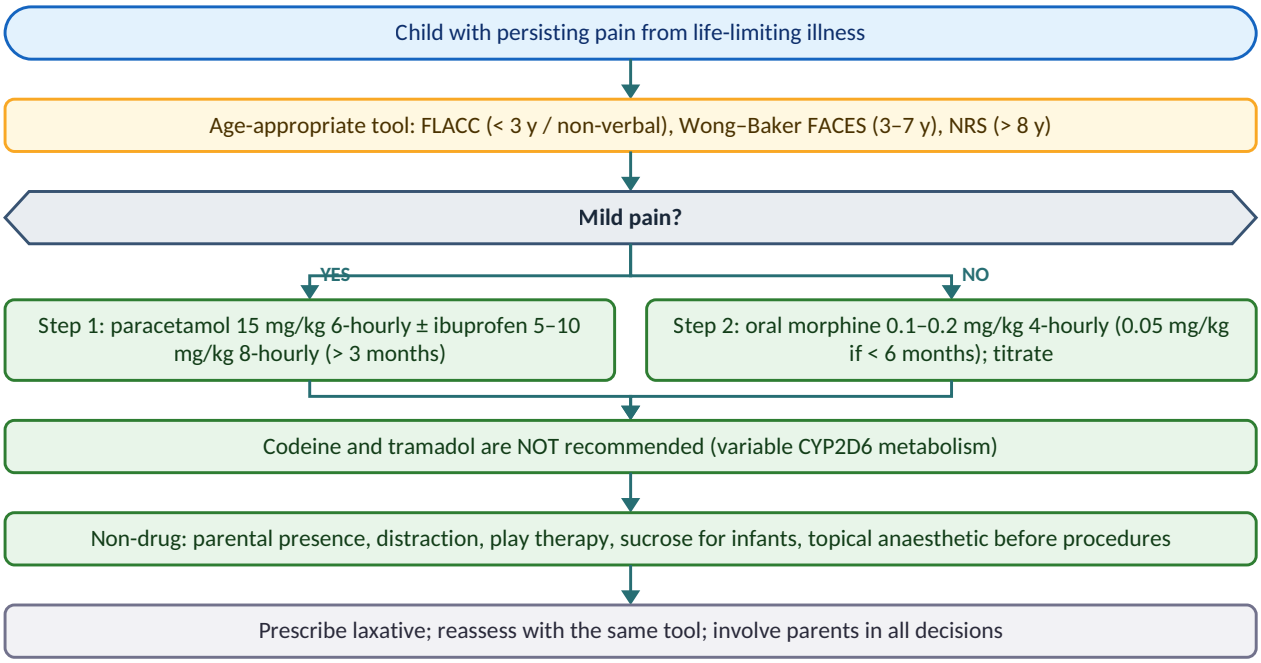
► In a non-communicative patient, a trial of analgesia ('analgesic trial') with reassessment of behavioural indicators is itself a valid diagnostic and therapeutic step – a commonly tested viva concept.

GUIDELINES & RECENT ADVANCES

- Warden V et al. Development and psychometric evaluation of the PAINAD scale. *J Am Med Dir Assoc* 2003

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ALGORITHM · Pain Management in Children Algo 32 — full page ›



★ **KEY POINTS**

- Children are not small adults — dose by weight and age.
- ACT categories 1–4 classify life-limiting conditions for paediatric palliative care.

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SECTION 3 · CHAPTER 3.3

Pharmacological Management

The pharmacological backbone of cancer and non-cancer pain control — the WHO analgesic ladder, opioid pharmacology, titration/rotation, adverse-effect management and adjuvant analgesia.

3.3.1 WHO Analgesic Ladder & Modifications 247

3.3.2 Opioid Pharmacology 248

3.3.3 Titration, Rotation & Breakthrough Pain 250

3.3.4 Opioid Adverse Effects & Toxicity 252

3.3.5 Non-Opioids & Adjuvant Analgesics 254

AT A GLANCE

Opioid essentials at a glance

Item	Rule
Starting dose (naive)	Oral morphine 2.5–5 mg 4-hourly
Rescue dose	1/6 of 24-h dose (= 4-h dose)
Titration	Increase by 30–50% using rescue use
Oral → SC morphine	Divide by 2
Renal failure	Fentanyl, buprenorphine, alfentanil
Always co-prescribe	Laxative; antiemetic PRN
Toxicity	Myoclonus, hallucinations, drowsiness — reduce / rotate



Freedom from cancer pain
Goal of the ladder — reassess regularly; step down when pain is controlled

4 **Refractory pain — interventional** ADDED 4TH STEP
Nerve blocks & neurolysis (coeliac plexus), intrathecal/epidural analgesia, palliative RT

3 **Step 3 · Severe pain (NRS 7–10) — strong opioid**
Morphine (first choice), fentanyl, buprenorphine, methadone **± non-opioid ± adjuvant**

2 **Step 2 · Moderate pain (NRS 4–6) — weak opioid**
Codeine or tramadol — or low-dose oral morphine (WHO 2018 / IAPC) **± non-opioid ± adjuvant**

1 **Step 1 · Mild pain (NRS 1–3) — non-opioid**
Paracetamol, NSAIDs **± adjuvant** (antidepressant, anticonvulsant, steroid, bisphosphonate, anxiolytic)

Foundation at every step — psychosocial & spiritual care
Explanation, listening to fears, family and social support, spiritual care, non-drug measures, treat the cause ("total pain")

Figure — WHO analgesic ladder (WHO 1986; IAPC, Twycross). By the mouth · by the clock · by the ladder · for the individual · attention to detail. Steps 1–3 are the WHO ladder; step 4 is a later addition. If pain persists, move up — not sideways within a group.

3.3.1 WHO Analgesic Ladder & Modifications

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** Describe the WHO three-step analgesic ladder with examples at each step. [Paper 2, every year]
- **Q** What modifications have been proposed to the classical WHO ladder? Discuss the 'fourth step'. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Master the three-step ladder and know when to bypass Step 2 for severe pain (CD2.1.2, CD2.1.3)
- ✓ Integrate the 'fourth step' (interventional techniques) into stepped care (CD9.3)

DEFINITION The WHO Analgesic Ladder (1986, updated 2018/2019) is a stepped framework for cancer pain relief: mild pain is treated with non-opioids ± adjuvants, moderate pain with weak opioids, and severe pain with strong opioids, titrated against response.

Step	Pain Severity	Drug Class
1	Mild (NRS 1–3)	Non-opioid (paracetamol/NSAID) ± adjuvant
2	Moderate (NRS 4–6)	Weak opioid (tramadol/codeine) + Step-1 drugs ± adjuvant
3	Severe (NRS 7–10)	Strong opioid (morphine and alternatives) + Step-1 drugs ± adjuvant
4 (modern addition)	Refractory to Step 3	Interventional techniques (nerve blocks, neuraxial analgesia, neurolysis, palliative RT)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Paracetamol	Step 1 — mild pain, antipyretic adjunct at every step	500–1000 mg PO/IV 6th hourly, max 3–4 g/day	Reduce max dose in hepatic impairment/ malnutrition/low body weight
Ibuprofen / Diclofenac (NSAID)	Step 1 — mild pain, especially bone/ inflammatory pain	Ibuprofen 400 mg PO 8th hourly (max 2.4 g/day)	Avoid in renal impairment, active GI bleed, uncontrolled heart failure; co-prescribe gastroprotection
Tramadol (Step 2)	Moderate pain	50–100 mg PO 6th hourly, max 400 mg/ day	Lowers seizure threshold; serotonin-syndrome risk with SSRIs/SNRIs/MAOIs
Morphine, immediate-release (Step 3)	Severe pain, cancer pain (WHO 2018/2019 guideline)	Start 5–10 mg PO 4th hourly in opioid-naïve patients; titrate to effect	Start lower and titrate slower in the elderly and in renal impairment

★ CLINICAL PEARLS & EXAM FOCUS

▸ WHO's 2018 adult cancer-pain guideline explicitly permits **bypassing Step 2** and starting a low-dose strong opioid directly for severe pain — a frequently-tested update to the 'classical' three-step teaching.

▸ Mnemonic: 'By the ladder, by the clock, by the mouth, by the individual, with attention to detail' (the five original WHO principles of cancer-pain relief).

GUIDELINES & RECENT ADVANCES

- WHO Guidelines for the pharmacological and radiotherapeutic management of cancer pain in adults and adolescents (2018)

3.3.2 Opioid Pharmacology

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Classify opioids. Describe the pharmacology, metabolism and adverse effects of morphine. [Paper 2, recurring]

Q Write a short note on opioid receptors and their clinical significance. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Describe opioid receptor pharmacology, classification and metabolism (CD2.1.2, CD2.1.3)

✓ Safely initiate and titrate opioids in a patient with cancer pain (PD3.6, PD3.8)

DEFINITION Opioids are agents that act on μ, κ and δ opioid receptors (G-protein-coupled, pre- and post-synaptic) to produce analgesia; classified by origin (natural/semi-synthetic/synthetic), by receptor activity (full agonist/ partial agonist/antagonist) and, on the WHO ladder, as weak (Step 2) or strong (Step 3).

Receptor	Principal Effect
μ (mu)	Supraspinal and spinal analgesia, euphoria, respiratory depression, constipation, dependence — the main analgesic target of clinical opioids
κ (kappa)	Spinal analgesia, sedation, dysphoria, diuresis
δ (delta)	Analgesia, mood modulation; minor role of most clinical opioids

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Codeine (weak, pro-drug)	Mild–moderate pain	30–60 mg PO 4–6th hourly, max 240 mg/day	Requires CYP2D6 conversion to morphine — unpredictable in poor/ ultra-rapid metabolisers; avoid in children
Tramadol (weak, mixed mechanism)	Mild–moderate pain	50–100 mg PO 6th hourly, max 400 mg/day	Also inhibits serotonin/ noradrenaline reuptake — serotonin-syndrome and seizure risk
Morphine (strong, gold standard)	Moderate–severe cancer pain	Oral IR 5–10 mg 4th hourly; SC dose = ½ of oral dose (IAPC) dose	Active metabolites (M6G, M3G) accumulate in renal failure — use with caution/ avoid in CKD
Fentanyl (strong, synthetic, lipophilic)	Stable pain, dysphagia, renal impairment	Transdermal patch 12–25 μg/h, changed 72-hourly (only once stabilised on an equivalent oral opioid dose)	Not for opioid-naïve or acute/ unstable pain; avoid external heat sources over the patch
Buprenorphine (strong, partial agonist)	Moderate–severe pain, renal impairment	Transdermal patch 5–20 μg/h weekly, or	Ceiling effect on respiratory depression (safer in overdose); can precipitate
		sublingual 200–400 μg 6–8th hourly	withdrawal if combined with a full agonist
Methadone (strong, NMDA antagonist)	Refractory/ neuropathic cancer pain, opioid rotation	Specialist-initiated only; highly variable half-life (8–60 h)	Risk of delayed accumulation, QT prolongation, and complex equianalgesic conversion — never initiate without experience

★ CLINICAL PEARLS & EXAM FOCUS

▸ Equianalgesic ratio (IAPC): oral : subcutaneous morphine = 2 : 1, i.e. the SC dose is one-half of the oral dose.

▸ Methadone conversion is **non-linear** – the oral-morphine-to-methadone ratio widens as the total morphine-equivalent dose increases; always convert under specialist/departmental protocol.

GUIDELINES & RECENT ADVANCES

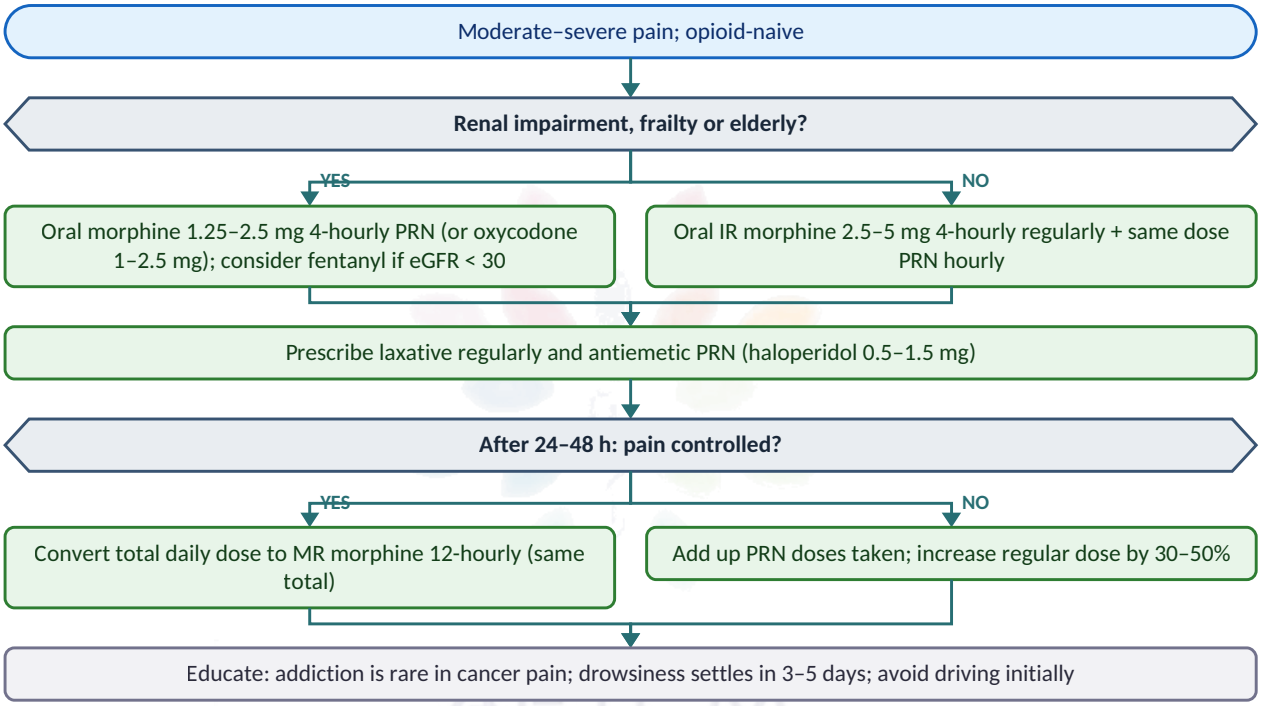
- WHO Model List of Essential Medicines (opioid analgesics section, most recent edition)

EXAM-ORIENTED THEORY

Opioids compared

Opioid	Oral morphine equivalence	Notes
Codeine	1/10	Prodrug (CYP2D6) — unpredictable
Tramadol	1/10	Serotonergic; seizures; renal caution
Tapentadol	1/2.5	MOR + noradrenaline reuptake inhibition
Oxycodone	1.5–2	Fewer hallucinations than morphine
Hydromorphone	5	Useful in renal impairment (with caution)
Fentanyl transdermal	12 µg/h ≈ 30 mg/24 h oral morphine	Renal-safe; for stable pain
Buprenorphine transdermal	10 µg/h ≈ 24 mg/24 h oral morphine	Renal-safe; ceiling for respiratory depression
Methadone	Variable (dose-dependent)	Specialist only; QTc

ALGORITHM · Starting Opioids in an Opioid-Naive Patient Algo 29 — full page ›



★ KEY POINTS

- ▸ Weak opioids (step 2) can be skipped — low-dose morphine is as effective (Bandieri 2016).
- ▸ Oral morphine bioavailability ~30%.

3.3.3 Titration, Rotation & Breakthrough Pain

Practice Qs › Algorithm ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** How do you titrate opioids in a patient with cancer pain? [Paper 2]
- **Q** What is opioid rotation? Describe the method of safe conversion between opioids. [Paper 2, frequently asked] Answer ›
- **Q** Define breakthrough pain. How is the rescue dose calculated? [Viva] Answer ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Calculate equianalgesic doses and perform safe opioid rotation (PD3.6)
- ✓ Prescribe appropriate rescue/breakthrough doses for incident and spontaneous pain (CD2.1.3)

DEFINITION **Titration** is the stepwise adjustment of regular (background) opioid dose to achieve analgesia with tolerable side-effects. **Opioid rotation** is switching from one opioid to another (usually for inadequate analgesia or intolerable toxicity), using equianalgesic conversion with an empirical dose-reduction for incomplete cross-tolerance. **Breakthrough pain** is a transient exacerbation of pain occurring despite adequate background analgesia.

Breakthrough pain — subtypes

Subtype	Feature
Spontaneous (idiopathic)	Unpredictable, no identifiable trigger
Incident (volitional)	Predictable, precipitated by a voluntary act, e.g. walking, dressing change
Incident (non-volitional)	Predictable, precipitated by an involuntary act, e.g. coughing
End-of-dose failure	Recurrence of pain before the next scheduled background dose is due — suggests the background dose/interval needs review, not just a rescue dose

Titration & rotation — key rules

- Titrate the total daily background dose upward by 30–50% if pain remains uncontrolled and the patient is using >3–4 rescue/breakthrough doses per day.
- Breakthrough ('rescue') dose = 1/6 of the total 24-hour opioid dose (= the 4-hourly dose, IAPC), as an immediate-release preparation.
- When rotating opioids: calculate the equianalgesic dose of the new opioid, then empirically reduce by 25–50% (incomplete cross-tolerance) before retitrating against response.

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Oral morphine IR (breakthrough)	Breakthrough/incident pain on a background oral-morphine regimen	1/6 of the total daily background dose, PRN	Space doses by at least 1 hour; review background dose if >4 rescue doses/day are needed
Sublingual/ transmucosal fentanyl	Rapid-onset breakthrough pain in an opioid-tolerant patient	Lowest available strength, titrate upward; onset 5– 15 min	Only in patients already opioid-tolerant (background morphine-equivalent ≥60 mg/ day); not interchangeable mcg-for-mcg between products

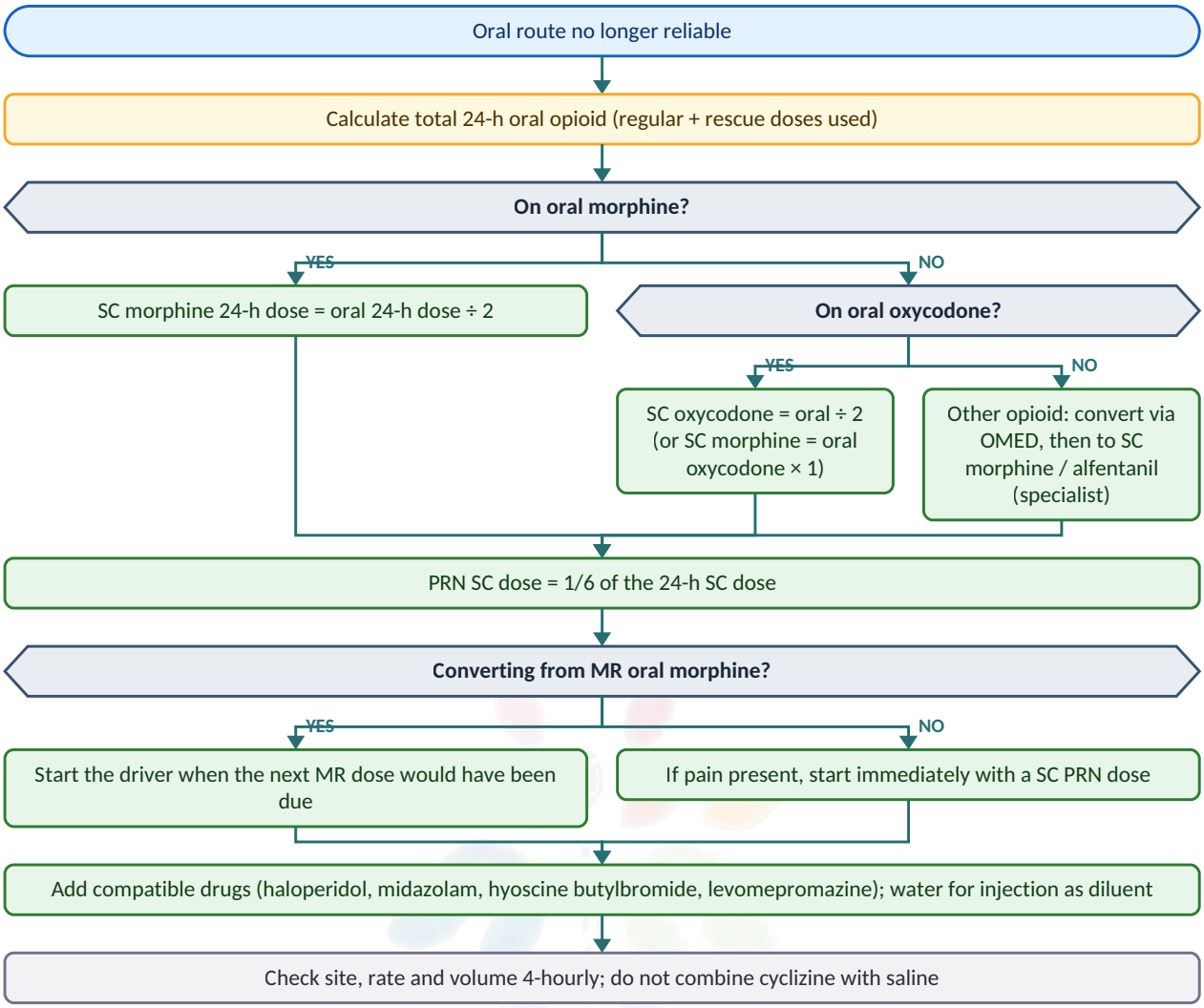
★ **CLINICAL PEARLS & EXAM FOCUS**

- ▸ Mnemonic for opioid rotation: 'Calculate → Reduce (25–50%) → Retitrate → Reassess'.
- ▸ Breakthrough analgesic requirement is itself a vital sign — persistently frequent rescue dosing is the earliest clue that the background regimen needs to change.

GUIDELINES & RECENT ADVANCES

- Davies A et al. EAPC recommendations on the management of breakthrough cancer pain. *Palliat Med* 2009

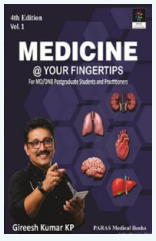
ALGORITHM · Converting to a Syringe Driver (CSCI) Algo 28 — full page ›



★ KEY POINTS

- ▸ Fentanyl patches should be continued, not converted, at end of life — add CSCI for extra needs.
- ▸ Driver takes ~4 h to reach steady state.

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3.3.4 Opioid Adverse Effects & Toxicity

Practice Qs ›
Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** Discuss the adverse effects of opioids and their management. [Paper 2]
- **Q** What is opioid-induced neurotoxicity? How would you manage opioid-induced respiratory depression? [Paper 2/Viva] Answer ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Prophylax and manage opioid-induced constipation, nausea, sedation, pruritus and myoclonus (CD2.1.2)
- ✓ Recognise and manage opioid-induced respiratory depression using naloxone protocols (PD3.6)

DEFINITION Opioid-induced neurotoxicity (OIN) is a syndrome of delirium, myoclonus, hyperalgesia and (rarely) seizures resulting from accumulation of opioids/active metabolites, most often in the context of renal impairment, dehydration or rapid dose escalation.

Adverse Effect	Typical Onset	First-Line Management
Constipation	Persists throughout treatment — no tolerance develops	Prophylactic stimulant ± softener laxative, started with every opioid prescription
Nausea/vomiting	First 3–5 days, usually tolerant thereafter	Short course of a dopamine antagonist (e.g. haloperidol/ metoclopramide)
Sedation	First 2–3 days	Usually self-limiting; review for over-rapid titration or accumulation
Pruritus	Variable	Antihistamine; consider opioid rotation if persistent
Myoclonus / OIN / hyperalgesia	Dose-related, especially in renal failure	Reduce dose, hydrate, rotate opioid; benzodiazepine for distressing myoclonus
Respiratory depression	Overdose / rapid accumulation	Stimulate/support airway; titrated naloxone (see below); consider the cause before resuming opioid

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Senna + docusate (stimulant + softener)	Prophylaxis/ treatment of opioid-induced constipation	Senna 15 mg PO at night, titrate; docusate 100 mg PO BD	Osmotic laxatives (lactulose) alone are often insufficient for opioid-induced constipation
Methylnaltrexone	Opioid-induced constipation refractory to standard laxatives	0.15 mg/kg subcutaneously alternate days (adult)	Avoid in known or suspected mechanical bowel obstruction
Haloperidol (low dose)	Opioid-induced nausea/ vomiting	0.5–1.5 mg PO/SC at night, titrate	QT prolongation at higher doses; caution in Parkinson's disease/Lewy body dementia
Naloxone			

Opioid-induced		0.04–0.1 mg IV boluses		Short half-life relative to
respiratory		every 2–3 min, titrated		most opioids — repeated
depression		to adequate respiration		dosing or an infusion is often
(reversal)		(not full arousal)		needed; precipitates acute
				withdrawal/pain crisis in an
				opioid-tolerant patient

★ CLINICAL PEARLS & EXAM FOCUS

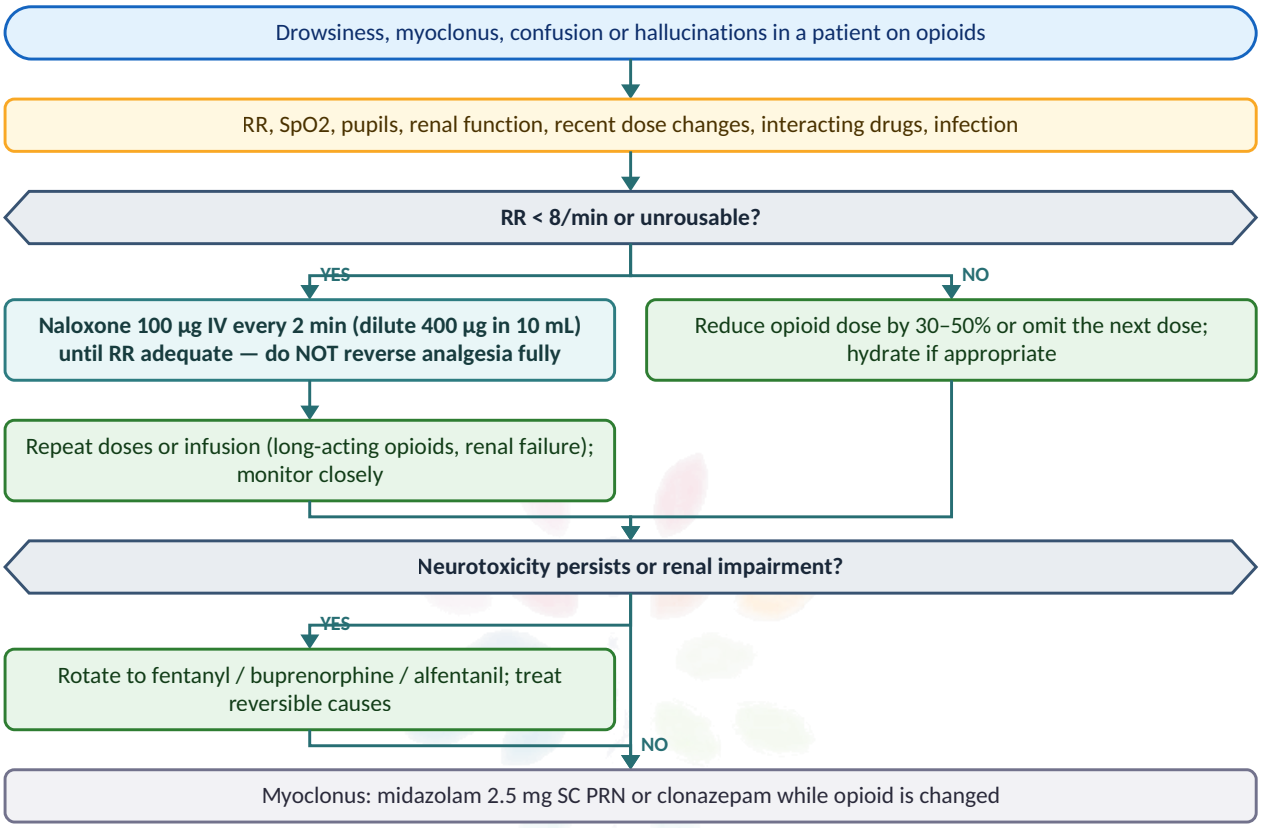
▸ Mnemonic for opioid adverse effects: 'SNOOP' – Sedation, Nausea, Obstipation (constipation), Overdose/ respiratory depression, Pruritus.

▸ Titrate naloxone to restore adequate respiration, *not* full consciousness – over-reversal precipitates an agonising acute pain crisis in a patient on long-term opioids for cancer pain.

GUIDELINES & RECENT ADVANCES

- Laugsand EA et al. Management of opioid-induced side effects in cancer patients: EAPC guidelines. *Palliat Med* 2011

ALGORITHM · Opioid Toxicity and Opioid-Induced Neurotoxicity Algo 24 — full page ›



★ KEY POINTS

▸ Full-dose naloxone in opioid-tolerant patients causes severe pain and withdrawal.

▸ Morphine-3- and -6-glucuronide accumulate in renal failure.

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3.3.5 Non-Opioids & Adjuvant Analgesics

Practice Qs ›
Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role of adjuvant analgesics in neuropathic cancer pain. [Paper 2, every year] Answer ›

Q Write short notes on the use of ketamine and corticosteroids in palliative pain management. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Evidence-based use of non-opioids and adjuvants — gabapentinoids, SNRIs/TCAs, corticosteroids, bisphosphonates, ketamine, lidocaine/capsaicin (CD2.1.4, CD2.1.5)

DEFINITION Adjuvant analgesics ('co-analgesics') are drugs whose primary indication is not pain, but which have an independent or synergistic analgesic effect in specific pain states — chiefly neuropathic and bone pain.

Class	Example	Best Evidence In
Gabapentinoid	Gabapentin, pregabalin	First-line neuropathic pain (CIPN, plexopathy)
SNRI / TCA	Duloxetine, amitriptyline	Neuropathic pain, especially with co-existing depression/sleep disturbance
Corticosteroid	Dexamethasone	Nerve-compression pain, raised intracranial pressure, bone pain, visceral capsular pain
NMDA antagonist	Ketamine	Refractory/severe neuropathic pain unresponsive to opioids and standard adjuvants
Bone-modifying agent	Zoledronic acid, denosumab	Metastatic bone pain, prevention of skeletal-related events
Topical agent	Lidocaine patch, capsaicin cream	Localised neuropathic/post-herpetic pain

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Gabapentin	Neuropathic pain (CIPN, plexopathy, post-herpetic neuralgia)	Start 100–300 mg PO at night, titrate to 300 mg TDS over days–weeks	Reduce dose in renal impairment; sedation, dizziness, ankle oedema
Amitriptyline	Neuropathic pain, co-existing insomnia	10–25 mg PO at night, titrate slowly	Anticholinergic effects, QT prolongation; caution in the elderly and cardiac disease
Dexamethasone	Nerve compression/cord compression, raised ICP, bone pain, anorexia	High-dose pulse (e.g. 16 mg/ day) in emergencies, tapering to lowest effective maintenance	Hyperglycaemia, proximal myopathy, GI bleeding, psychiatric effects, oral candidiasis with prolonged use
Ketamine	Refractory neuropathic/ opioid-poorly-responsive pain	Subcutaneous infusion, specialist-initiated, low starting dose titrated to effect	Dissociative/psychomimetic effects, hypertension; requires experienced supervision
Zoledronic acid	Metastatic bone pain, hypercalcaemia of malignancy	4 mg IV over ≥15 min, every 3– 4 weeks	Renal toxicity (monitor creatinine); risk of osteonecrosis of the jaw with prolonged use

★ CLINICAL PEARLS & EXAM FOCUS

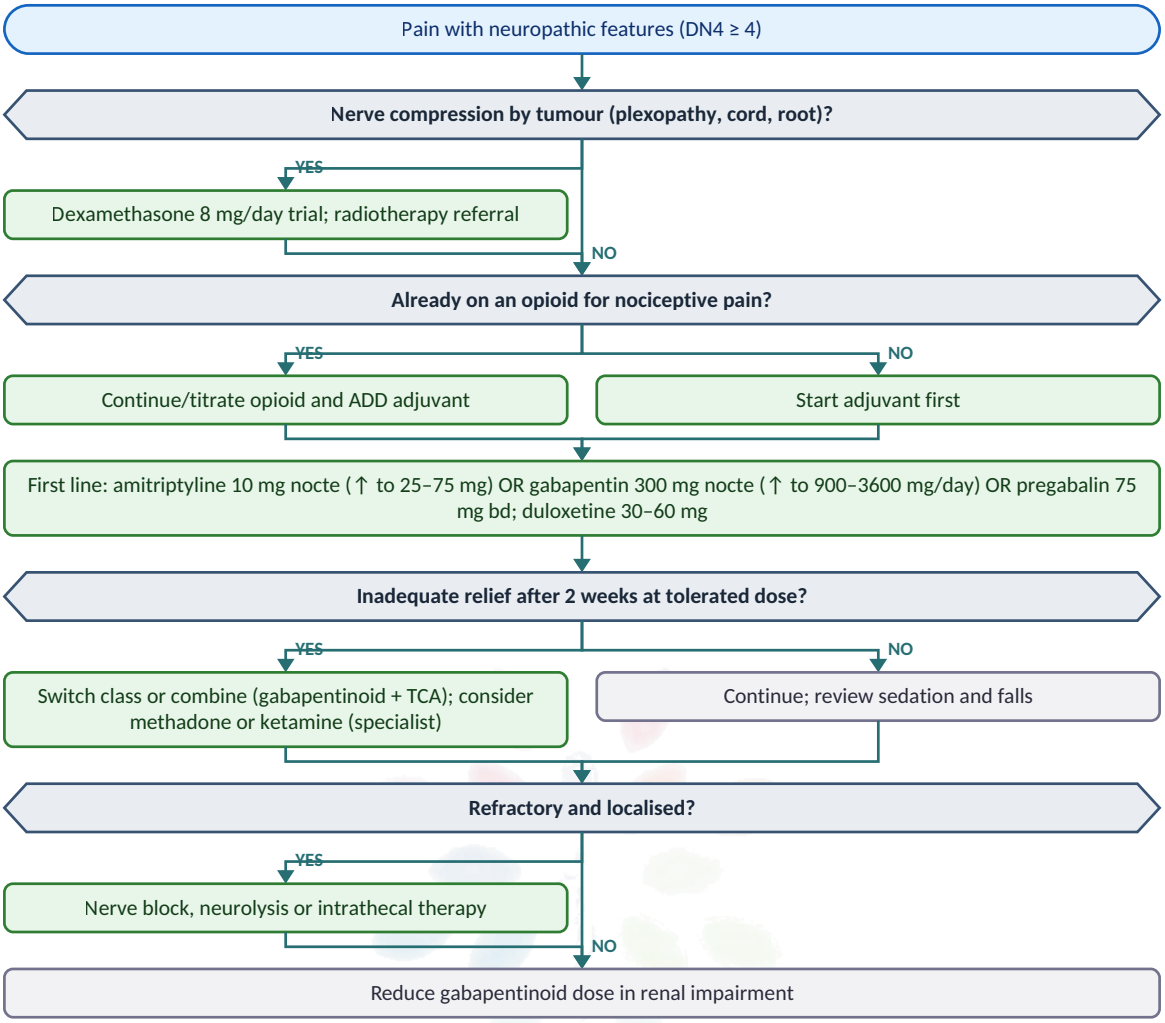
▸ The 'Neuropathic Pain Step Ladder' (first-line gabapentinoid/TCA/SNRI → combination of two classes → refer for interventional/ketamine) is a favourite short-note topic.

▸ Dexamethasone is the single most versatile adjuvant in palliative medicine – it treats pain, nausea, anorexia and raised ICP simultaneously, which is exactly why examiners like to test it.

GUIDELINES & RECENT ADVANCES

- Finnerup NB et al. Pharmacotherapy for neuropathic pain in adults: a systematic review and meta-analysis (NeuPSIG). *Lancet Neurol* 2015

ALGORITHM · Neuropathic Pain Pharmacotherapy Algo 25 — full page ›



★ KEY POINTS

- ▸ Number needed to treat ~3–4 for TCAs; ~6–8 for gabapentinoids.
- ▸ Amitriptyline: caution in cardiac disease and elderly.

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SECTION 3 · CHAPTER 3.4

Interventional & Disease-Modifying Therapies

The 'fourth step' of the analgesic ladder — nerve blocks, neuraxial analgesia and oncologic interventions for pain refractory to systemic pharmacotherapy.

3.4.1 Sympathetic & Peripheral Nerve Blocks

3.4.2 Neuraxial Analgesia **258**

3.4.3 Palliative Oncologic Interventions **259**

AT A GLANCE

Interventions at a glance

Technique	Indication
Coeliac plexus block	Pancreatic / upper abdominal pain
Superior hypogastric / ganglion impar	Pelvic / perineal pain
Intrathecal pump	Refractory diffuse pain, prognosis > 3 months
Cordotomy	Unilateral pain below C5 (mesothelioma)
Vertebroplasty / kyphoplasty	Painful vertebral fractures
Radiotherapy 8 Gy single fraction	Bone metastasis pain

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3.4.1 Sympathetic & Peripheral Nerve Blocks

Practice Qs ›
Algorithm ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the indications, technique and complications of a coeliac plexus block. [Paper 2]

Q Write short notes on the superior hypogastric plexus block and ganglion impar block. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Describe indications, anatomy and complications of coeliac plexus, superior hypogastric plexus, ganglion impar and stellate/intercostal blocks (CD9.3.1)

DEFINITION Sympathetic and peripheral nerve blocks are interventional procedures that interrupt nociceptive and sympathetic afferent pathways using local anaesthetic (diagnostic/prognostic block) or a neurolytic agent (phenol/alcohol — therapeutic block), reserved for visceral or regional pain refractory to systemic therapy.

Block	Indication	Key Complication
Coeliac plexus block	Pancreatic and upper GI cancer pain	Orthostatic hypotension, diarrhoea, retroperitoneal haemorrhage, (rare) paraplegia
Superior hypogastric plexus block	Pelvic malignancy pain (cervix, rectum, bladder, prostate)	Vascular/nerve injury, bowel/bladder dysfunction (rare)
Ganglion impar block	Perineal/anorectal cancer pain	Rectal perforation (rare), transient numbness
Stellate ganglion block	Upper-limb complex regional pain, head/neck sympathetically-maintained pain	Horner's syndrome (expected), hoarseness, pneumothorax
Intercostal nerve block	Rib metastasis, post-thoracotomy pain	Pneumothorax, local anaesthetic systemic toxicity

Drugs used — Drug · Indication · Dose · Caution

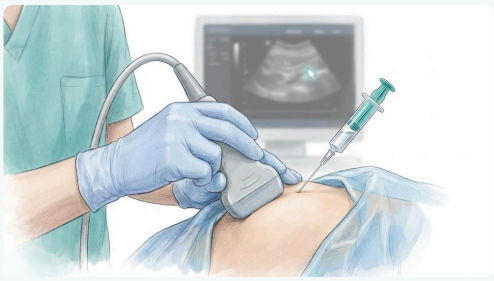
Drug	Indication	Dose	Caution
Bupivacaine 0.25–0.5%	Diagnostic/prognostic local anaesthetic block before neurolysis	Volume per block site (e.g. 10–20 mL for coeliac plexus)	Local anaesthetic systemic toxicity with inadvertent intravascular injection
Absolute alcohol (50–100%)	Neurolytic coeliac plexus/ splanchnic block	Per procedure, image-guided	Neuritis, deafferentation pain if misplaced
Phenol (6–10% aqueous)	Neurolytic block where alcohol-related dysesthesia is a concern	Per procedure, image-guided	Less painful on injection than alcohol but similar neurolytic risk profile

★ CLINICAL PEARLS & EXAM FOCUS

► A coeliac plexus neurolytic block gives good-to-excellent pain relief in roughly 70–90% of pancreatic cancer patients – a frequently quoted statistic in viva.

GUIDELINES & RECENT ADVANCES

- Mercadante S. Neuraxial techniques for cancer pain: an opinion paper. *Eur J Pain* 2001 (classical reference, freely indexed)



ILLUSTRATION

Image-guided nerve block

- Ultrasound, fluoroscopy or CT guidance improves accuracy and safety.
- A diagnostic local-anaesthetic block comes before neurolysis (alcohol or phenol).
- Check coagulation and consent; after coeliac block watch for postural hypotension and diarrhoea.

3.4.2 Neuraxial Analgesia

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the indications and management of continuous epidural/intrathecal analgesia in cancer pain. [Paper 2]

Q What drugs are used in intrathecal analgesia? Discuss early and late complications. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✔ Manage continuous epidural and intrathecal drug-delivery systems for refractory pain (CD9.3.1, PD3.10)

DEFINITION Neuraxial analgesia delivers analgesic/adjuvant drugs directly into the epidural or intrathecal (subarachnoid) space, achieving effective analgesia at a fraction of the systemic dose and side-effect burden, for pain refractory to optimised systemic opioid/adjuvant therapy.

Feature	Epidural	Intrathecal
Site	Epidural space	Subarachnoid (CSF) space
Relative potency	Lower (drug must cross dura)	10× more potent than epidural; 100× more than systemic morphine
Typical duration of use	Shorter-term (catheter migration/infection risk over weeks)	Longer-term, including implanted pump systems
Common early complications	Catheter migration, local infection, hypotension (with local anaesthetic)	Post-dural-puncture headache, infection (meningitis)
Common late complications	Catheter fibrosis/occlusion	Granuloma formation at catheter tip (especially with high-dose morphine)

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Morphine (preservative-free)	Neuraxial analgesia, refractory cancer pain	Intrathecal dose ≈ 1/10 of epidural dose ≈ 1/100 of oral dose	Delayed respiratory depression (rostral spread); monitor closely after dose changes
Bupivacaine (low concentration)	Added for incident/ neuropathic pain not controlled by opioid alone	Low concentration (e.g. 0.1–0.25%) by continuous infusion	Motor block, hypotension at higher concentrations
Clonidine	Adjuvant for neuropathic pain component	Low-dose continuous infusion, specialist-titrated	Hypotension, sedation
Ziconotide	Refractory cancer pain unresponsive to opioid-based neuraxial regimens (specialist/ tertiary-centre use)	Specialist-initiated, very low starting dose, slow titration	Neuropsychiatric adverse effects (confusion, hallucinations); not an opioid — no respiratory depression but narrow therapeutic window

★ CLINICAL PEARLS & EXAM FOCUS

▸ Memorise the potency ratio: oral morphine : parenteral morphine : epidural morphine : intrathecal morphine ≈ 300 : 100 : 10 : 1 (approximate teaching ratio).

GUIDELINES & RECENT ADVANCES

- Deer TR et al. The Polyanalgesic Consensus Conference (PACC) guidelines for intrathecal drug delivery

3.4.3 Palliative Oncologic Interventions

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role of palliative radiotherapy in bone pain. [Paper 2]

Q Write short notes on radiopharmaceuticals used in metastatic bone pain. [Viva]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Use single-fraction/hypofractionated palliative RT, radiopharmaceuticals and orthopaedic stabilisation for pain control (CD4.1.6, CD4.3)

DEFINITION Palliative oncologic interventions use disease-directed modalities — radiotherapy, radiopharmaceuticals and orthopaedic stabilisation — specifically to control pain and prevent skeletal complications, rather than to achieve cure.

Modality	Typical Use	Key Point
Single-fraction palliative RT (e.g. 8 Gy × 1)	Uncomplicated painful bone metastasis	Equivalent pain-control to multi-fraction regimens with less patient burden; slightly higher retreatment rate
Hypofractionated RT (e.g. 20 Gy/5#, 30 Gy/10#)	Complicated bone metastasis (impending fracture, soft-tissue mass, prior RT field)	Chosen when durability of response and bone re-mineralisation are priorities
Radiopharmaceuticals (Strontium-89, Samarium-153)	Widespread painful osteoblastic bone metastases (e.g. prostate cancer)	Systemic, repeatable; myelosuppression is the dose-limiting toxicity
Prophylactic orthopaedic fixation	Impending pathological fracture (Mirel's score ≥8)	Prevents a fracture and its associated pain crisis; done before, not after, a fracture where possible

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Strontium-89 chloride	Painful, widespread osteoblastic bone metastases	Single IV dose, specialist nuclear-medicine administration	Myelosuppression (nadir 4–6 weeks); avoid if platelet/white-cell count already low
Samarium-153 EDTMP	Painful osteoblastic bone metastases	Single IV dose, weight-based, specialist administration	Myelosuppression; transient pain flare in the first 48–72 h
Zoledronic acid / Denosumab	Adjunct to reduce skeletal-related events and bone pain	Zoledronic acid 4 mg IV 3–4 weekly; denosumab 120 mg SC 4-weekly	Monitor renal function (zoledronic acid) and calcium (denosumab); osteonecrosis of the jaw risk with prolonged use

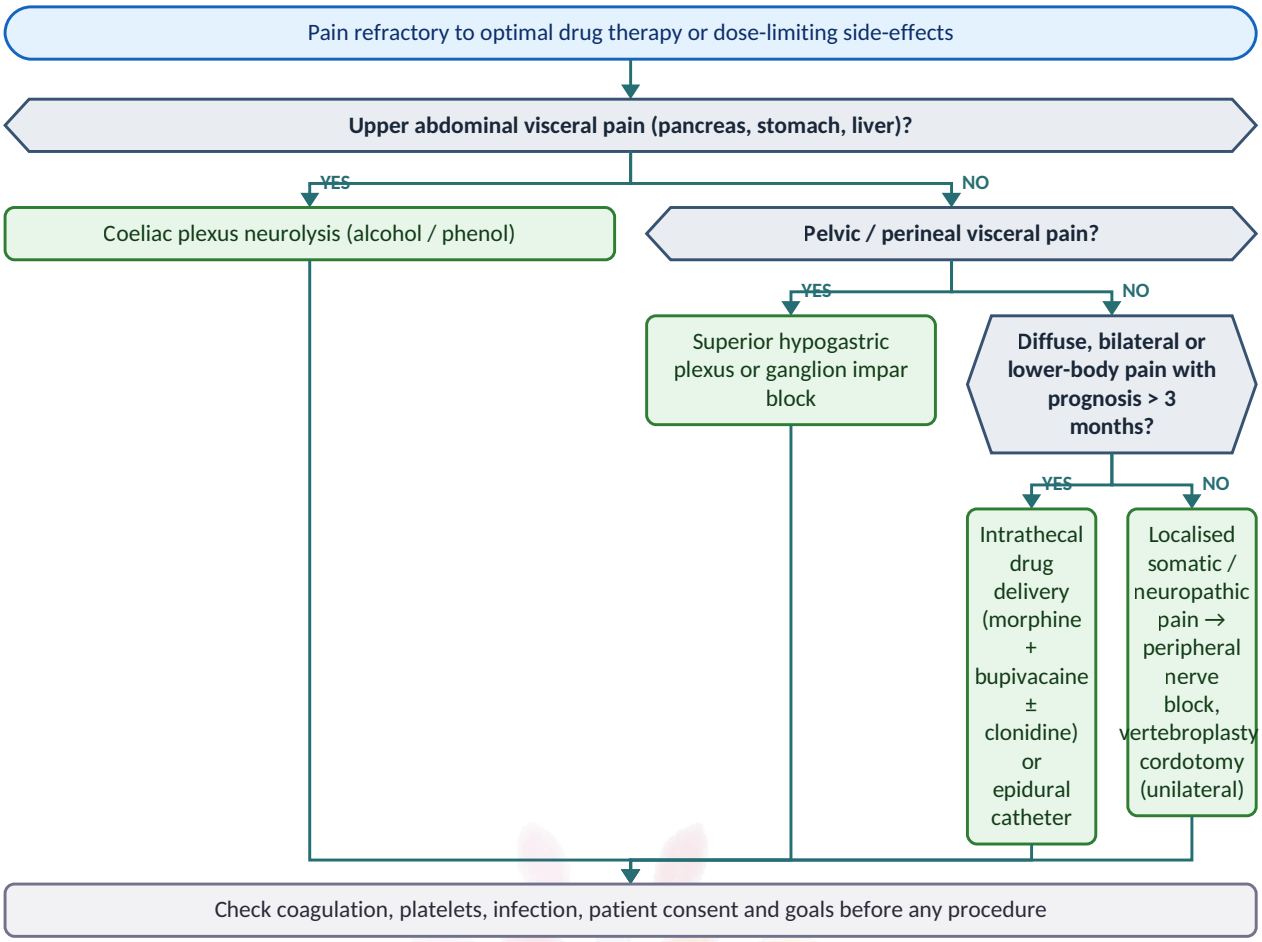
★ CLINICAL PEARLS & EXAM FOCUS

► Mirel's scoring system (site, size, nature of lesion, pain) predicts pathological-fracture risk; a score of ≥8/12 is the standard threshold for prophylactic fixation – a classic viva number to remember.

GUIDELINES & RECENT ADVANCES

- Lutz S et al. Palliative radiation therapy for bone metastases: ASTRO evidence-based guideline. *Int J Radiat Oncol Biol Phys* 2011

ALGORITHM · Choosing an Interventional Pain Technique Algo 46 — full page ›



★ **KEY POINTS**

- Early coeliac block reduces opioid use and constipation in pancreatic cancer.
- Percutaneous cervical cordotomy suits unilateral mesothelioma pain.

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SECTION 3 · CHAPTER 3.5

Pain Emergencies & Complex Scenarios

Time-critical oncologic pain crises, dosing in organ failure, and the stewardship of opioids in patients with substance-use vulnerability.

3.5.1 Oncologic Pain Crises 262

3.5.2 Organ Failure Dosing 263

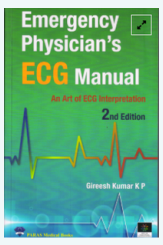
3.5.3 Substance Use & Opioid Stewardship 264

AT A GLANCE

Pain emergencies at a glance

Emergency	First action
Pain crisis	IV/SC opioid titration every 15 min
Spinal cord compression	Dexamethasone 16 mg; MRI within 24 h
Pathological fracture	Splint, analgesia, orthopaedic review
Bowel perforation / obstruction	Surgical review vs comfort care
Opioid toxicity	Titrated naloxone, dose reduction

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3.5.1 Oncologic Pain Crises

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** Discuss the emergency management of malignant spinal cord compression. [Paper 2/4, every year] Answer ›
- **Q** Outline your approach to a patient with sudden severe abdominal pain on a background of advanced ovarian cancer. [Paper 2]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Rapidly recognise and manage malignant spinal cord compression, pathological fracture, visceral perforation and acute bowel obstruction (CD4.3)

DEFINITION An oncologic pain crisis is a sudden, severe exacerbation of pain signalling a potentially reversible, time-critical complication (cord compression, fracture, perforation, obstruction) that requires emergency assessment alongside — not instead of — immediate analgesia.

Crisis	Red-Flag Clue	Immediate Action
Malignant spinal cord compression	Severe back pain + limb weakness/ sensory level/sphincter disturbance	High-dose dexamethasone immediately, urgent whole-spine MRI, urgent oncology/neurosurgery referral
Pathological fracture (impending/actual)	Severe localised bone pain, worse on weight-bearing, Mirel's score ≥8	Immobilise/analgese, urgent orthopaedic referral for fixation
Visceral perforation / peritonitis	Sudden severe abdominal pain with guarding, fever	Analgesia does not await surgical/medical assessment; urgent imaging and surgical opinion within goals-of-care
Acute malignant bowel obstruction	Colicky abdominal pain, vomiting, absolute constipation	Opioid + antispasmodic (hyoscine) + antiemetic; assess suitability for surgery/stenting vs conservative management

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Dexamethasone (high-dose)	Malignant spinal cord compression / raised intracranial pressure	16 mg IV/PO stat, then 8 mg BD, tapering once definitive treatment (RT/ surgery) planned	Hyperglycaemia, GI bleeding, psychiatric effects — still outweighed by urgency of neurological preservation
Morphine (parenteral)	Severe pain crisis of any oncologic emergency	2.5–5 mg IV/SC, repeated every 10–15 min, titrated to effect	Do not let concern about respiratory depression delay adequate analgesia in a crisis — titrate carefully and reassess
Hyoscine butylbromide	Colicky pain of malignant bowel obstruction	20 mg SC/IV QDS, up to 120 mg/24 h by CSCI	Does not cross the blood–brain barrier (unlike hyoscine hydrobromide) — no sedative/ confusional effect

★ CLINICAL PEARLS & EXAM FOCUS

▸ "Back pain + leg weakness in a cancer patient is spinal cord compression until proven otherwise" – this single sentence, correctly actioned, is worth disproportionate marks in both theory and OSCE stations.

▸ Adequate analgesia must never be delayed while awaiting a definitive diagnosis in a pain crisis — treat the pain and investigate the cause in parallel.

GUIDELINES & RECENT ADVANCES

- Loblaw DA et al. Malignant extradural spinal cord compression: ASCO clinical practice guideline

3.5.2 Organ Failure Dosing

Practice Qs › Algorithm › NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** How would you prescribe opioids for pain in a patient with end-stage renal disease? [Paper 3] Answer ›
- **Q** Discuss analgesic dose adjustment in hepatic failure. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Adjust analgesics appropriately in severe renal and hepatic impairment (CD2.7.1)

DEFINITION Organ-failure dosing is the deliberate selection and dose-adjustment of analgesics to avoid toxic accumulation of parent drug or active metabolites in patients with significantly impaired renal or hepatic clearance.

Organ Failure	Preferred Opioid(S)	Opioids To Avoid/Use With Caution
Severe renal impairment / ESRD	Fentanyl, buprenorphine (no active renal-excreted metabolites); methadone (specialist use)	Morphine and codeine — active metabolites (M6G, M3G) accumulate and cause toxicity/ OIN
Severe hepatic impairment	Fentanyl (still needs caution — reduced clearance in severe disease); lower starting doses of most opioids with slower titration	Avoid long-acting/sustained-release formulations until response to short-acting agents is established

Adjuvant	Renal Adjustment	Hepatic Adjustment
Gabapentin/ pregabalin	Significant dose reduction required (renally cleared)	No major adjustment usually required
NSAIDs	Avoid — risk of acute kidney injury	Avoid in significant hepatic synthetic dysfunction (bleeding risk)
Paracetamol	No adjustment usually needed	Reduce maximum daily dose in significant impairment/ malnutrition

Drugs used — Drug · Indication · Dose · Caution

Drug	Indication	Dose	Caution
Fentanyl	Strong-opioid analgesia in renal impairment	Transdermal patch 12–25 µg/h once stabilised on an equivalent oral opioid	Caution in severe hepatic impairment — reduced clearance; avoid in opioid-naïve/ unstable pain
Buprenorphine	Strong-opioid analgesia in renal and mild–moderate hepatic impairment	Transdermal 5–20 µg/h weekly, or sublingual 6– 8th hourly	Ceiling effect on respiratory depression; partial agonist — avoid combining with a full agonist
Methadone	Refractory pain in renal impairment under specialist supervision	Specialist-initiated only	Hepatically metabolised with minimal renal excretion, but complex pharmacokinetics — never a first-line choice outside a specialist service

★ CLINICAL PEARLS & EXAM FOCUS

▸ Mnemonic: in renal failure, think 'F-B-M' – Fentanyl, Buprenorphine, Methadone – the three opioids of choice, in roughly that order of ease of use.

GUIDELINES & RECENT ADVANCES

- King S et al. A systematic review of the use of opioid medication for those with moderate to severe renal impairment: EAPC. *Palliat Med* 2011

3.5.3 Substance Use & Opioid Stewardship

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Differentiate tolerance, dependence, pseudoaddiction and opioid use disorder. [Paper 2/Viva]

Q Discuss the regulatory framework governing opioid prescribing in India. [Paper 1/4]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Differentiate physical dependence, tolerance, pseudoaddiction and opioid use disorder (AD2.3)

✓ Navigate regulatory frameworks such as the NDPS Act for essential narcotic drugs (CD1.9.1)

DEFINITION **Tolerance** is the need for an increasing dose to maintain the same effect. **Physical dependence** is the occurrence of a withdrawal syndrome on abrupt cessation — both are expected, predictable physiological phenomena of long-term opioid use and are *not* addiction. **Pseudoaddiction** is drug-seeking behaviour that results from undertreated pain and resolves once pain is adequately controlled. **Opioid use disorder** is a pattern of compulsive use with loss of control despite harm — a distinct psychiatric diagnosis.

Term	Key Distinguishing Feature
Tolerance	Need for escalating dose for the same analgesic effect — physiological, expected
Physical dependence	Withdrawal syndrome on abrupt stop/rapid dose reduction — physiological, expected
Pseudoaddiction	Drug-seeking that resolves promptly once pain is adequately treated — a sign of *under*-treatment
Opioid use disorder (addiction)	Compulsive use, loss of control, continued use despite harm — a distinct, less common psychiatric diagnosis in the cancer-pain population

Regulatory framework in India

The Narcotic Drugs and Psychotropic Substances (NDPS) Act, 1985, as amended in 2014, introduced the category of "Essential Narcotic Drugs" (including oral morphine) and simplified multi-state licensing to a single recognised-medical-institution licence, substantially improving opioid availability for legitimate pain management across India.

★ CLINICAL PEARLS & EXAM FOCUS

▸ A rising analgesic requirement in a patient with advanced cancer is overwhelmingly more likely to be disease progression or pseudoaddiction than true opioid use disorder — do not under-treat pain out of an unfounded fear of 'addiction'.

▸ The 2014 NDPS amendment is one of the most frequently tested policy facts in Paper 1/4 — know the year and the term 'Essential Narcotic Drug'.

GUIDELINES & RECENT ADVANCES

- Rajagopal MR, Joranson DE. India: opioid availability — an update. *J Pain Symptom Manage* 2007; NDPS (Amendment) Act, 2014

EXAM-ORIENTED THEORY

Opioid stewardship in palliative practice

Element	Practice
Risk screening	Opioid Risk Tool (ORT), CAGE-AID, history of substance use
Agreement	Single prescriber, single pharmacy, written plan
Formulation	Long-acting preparations; limited quantities
Monitoring	Pill counts, urine drug screen with consent, functional goals
Safety	Naloxone education for families; safe storage and disposal
India	NDPS Amendment 2014; Recognised Medical Institutions (RMIs) for morphine dispensing

SECTION 3 · CHAPTER 3.6

Non-Pharmacological & Rehabilitative Approaches

Physical, rehabilitative, psychological and integrative modalities that complement pharmacotherapy and address the non-physical dimensions of total pain.

3.6.1 Physical & Rehabilitative Modalities **266**

3.6.2 Psychological & Integrative Therapies **267**

AT A GLANCE

Non-drug approaches at a glance

Approach	Example
Physical	TENS, heat/cold, positioning, exercise, massage
Psychological	CBT, relaxation, mindfulness, hypnosis
Rehabilitative	Physiotherapy, occupational therapy, aids
Integrative	Yoga, music therapy, acupuncture
Spiritual	Meaning-centred and dignity therapy

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3.6.1 Physical & Rehabilitative Modalities

Practice Qs ›
NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role of physiotherapy and TENS in palliative pain management. [Paper 3]

Q Describe complete decongestive therapy for cancer-associated lymphoedema. [Paper 3]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply physiotherapy, lymphoedema management, orthotics, TENS and positioning strategies (CD10.2, PD8.9)

DEFINITION Non-pharmacological physical modalities use movement, positioning, external devices or neuromodulation to reduce pain and preserve function, and are used alongside — never instead of — indicated pharmacotherapy.

Modality	Typical Indication	Key Point
Physiotherapy / graded exercise	Deconditioning, musculoskeletal pain, maintaining mobility	Improves function and may reduce analgesic requirement; tailored to prognosis and tolerance
TENS (transcutaneous electrical nerve stimulation)	Localised musculoskeletal/ neuropathic pain	Low-risk adjunct; evidence modest but widely used as a low-cost option
Complete Decongestive Therapy (CDT)	Cancer-associated lymphoedema with associated heaviness/pain	Manual lymphatic drainage + compression bandaging/ garments + exercise + skin care, in intensive then maintenance phases
Orthotics / positioning	Bone pain, impending fracture, pressure areas	Splinting, supportive positioning and pressure redistribution reduce incident pain and prevent complications

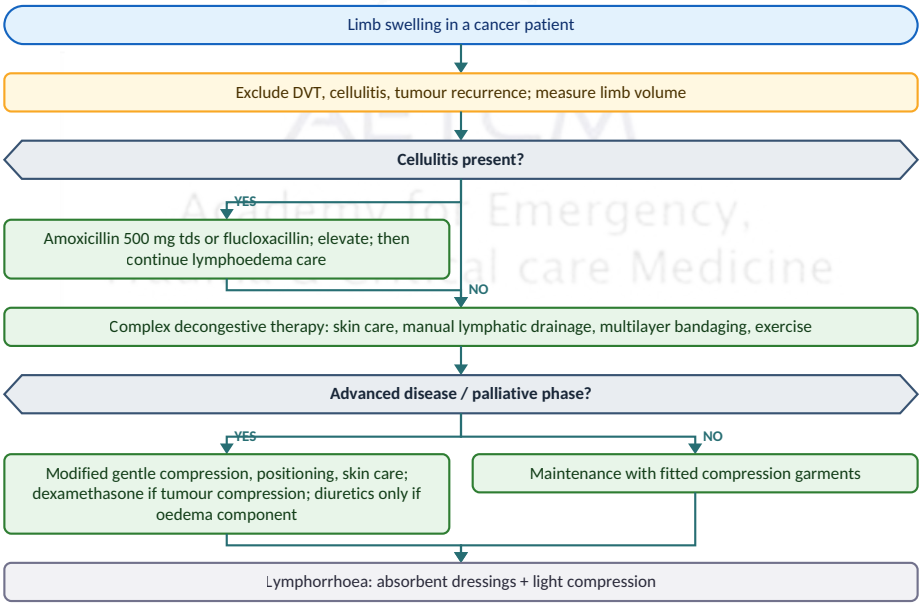
★ CLINICAL PEARLS & EXAM FOCUS

▸ CDT has two phases – an intensive reduction phase and a lifelong maintenance phase – a distinction commonly tested in short-answer questions.

GUIDELINES & RECENT ADVANCES

- International Lymphoedema Framework / ISL consensus documents on lymphoedema management

ALGORITHM · Lymphoedema Management Algo 51 — full page ›



3.6.2 Psychological & Integrative Therapies

NMC curriculum ›

PREVIOUS-YEAR-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss psychological interventions for pain in a patient with advanced cancer. [Paper 3]

Q What is the role of integrative medicine in palliative pain management? [Paper 3, newer theme]

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Apply cognitive-behavioural therapy, mindfulness, guided imagery, music therapy and spiritual counselling to address total suffering (CD9.4, AD4)

DEFINITION Psychological and integrative therapies target the cognitive, emotional, social and spiritual components of total pain, reducing pain-related distress and amplification even when the nociceptive stimulus itself cannot be fully eliminated.

Therapy	Mechanism / Use
Cognitive-behavioural therapy (CBT)	Restructures catastrophising thoughts about pain; builds coping and self-efficacy
Mindfulness-based stress reduction	Reduces the affective amplification of pain through present-moment, non-judgemental awareness
Guided imagery / relaxation	Reduces muscle tension and sympathetic arousal associated with pain
Music therapy	Reduces anxiety and pain perception; evidence strongest for procedural and anticipatory pain/anxiety
Spiritual counselling / chaplaincy	Addresses the spiritual/existential component of total pain (meaning, hope, reconciliation)

Integrative Medicine in Palliative Medicine (CD9.4)

Integrative approaches (yoga, acupuncture/acupressure, selected botanical preparations) are used as adjuncts within a structured evidence-based framework, always disclosed to and coordinated with the primary treating team to avoid interaction with disease-modifying or analgesic therapy.

★ CLINICAL PEARLS & EXAM FOCUS

► In an examination answer on 'holistic pain management', always explicitly link each non-pharmacological modality back to one of the four domains of total pain (physical/psychological/social/spiritual) – this framing is what separates a pass-level answer from a distinction-level one.

GUIDELINES & RECENT ADVANCES

- Mao JJ et al. Integrative oncology for pain management: SIO-ASCO guideline. *J Clin Oncol* 2022

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SECTION 4

Question & Answer Practice

Comprehensive exam-preparatory practice drawing on both the CBME Curriculum (Part I) and Pain Management (Section 3 (Pain Management)) — an Algorithm Atlas, Solved Clinical Case Scenarios, Model Long-Essay Answers, Short-Essay Model Answers, Viva Voce Rapid-Fire, the MCQ Question Bank, a Landmark Trials reference, and an official DNB previous-year question archive.

1 Algorithm Atlas

2 Solved Clinical Case Scenarios

3 Model Long-Essay Answers

4 Short-Essay Model Answers

5 Viva Voce Rapid-Fire

6 MCQ Question Bank

7 Landmark Trials in Palliative Medicine & Oncology **423**

8 DNB Previous-Year Question Papers (Official NBE Archive) **427**

Algo Algorithm Atlas (61) **269**

Case Solved Clinical Cases (13) **330**

LE Model Long-Essay Answers (15) **344**

SE Model Short-Essay Answers (29) **359**

Viva Viva Voce Rapid-Fire (10) **389**

MCQ MCQ Question Bank (26) **399**

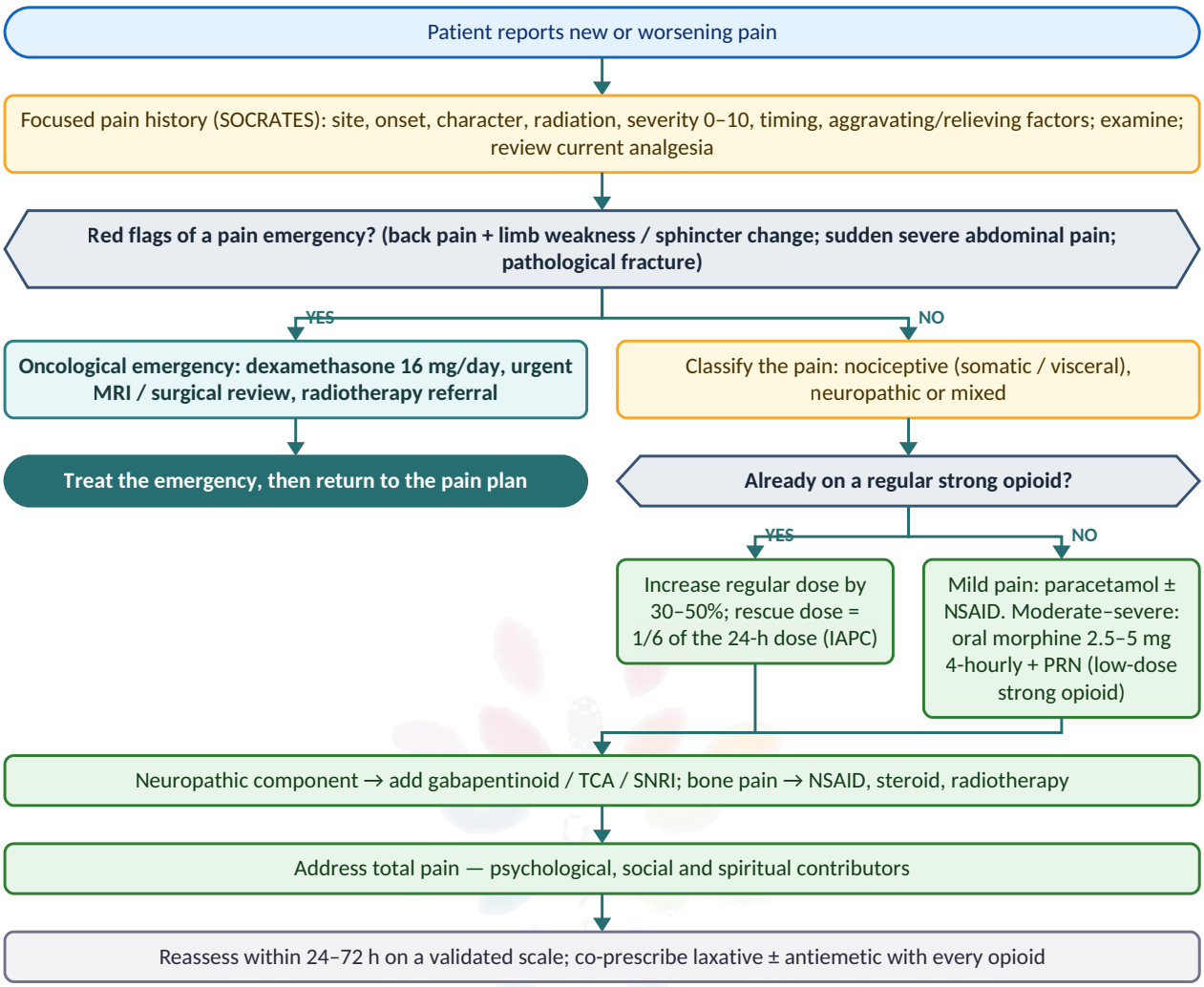
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ALGORITHM ATLAS · Algo 1

Algorithm: Approach to New or Worsening Pain in a Palliative Care Patient

Read the text ›

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Always exclude a pain emergency first — spinal cord compression and pathological fracture are time-critical.
- Titrate by 30–50% steps; 'by the clock, by the mouth, by the ladder, for the individual'.

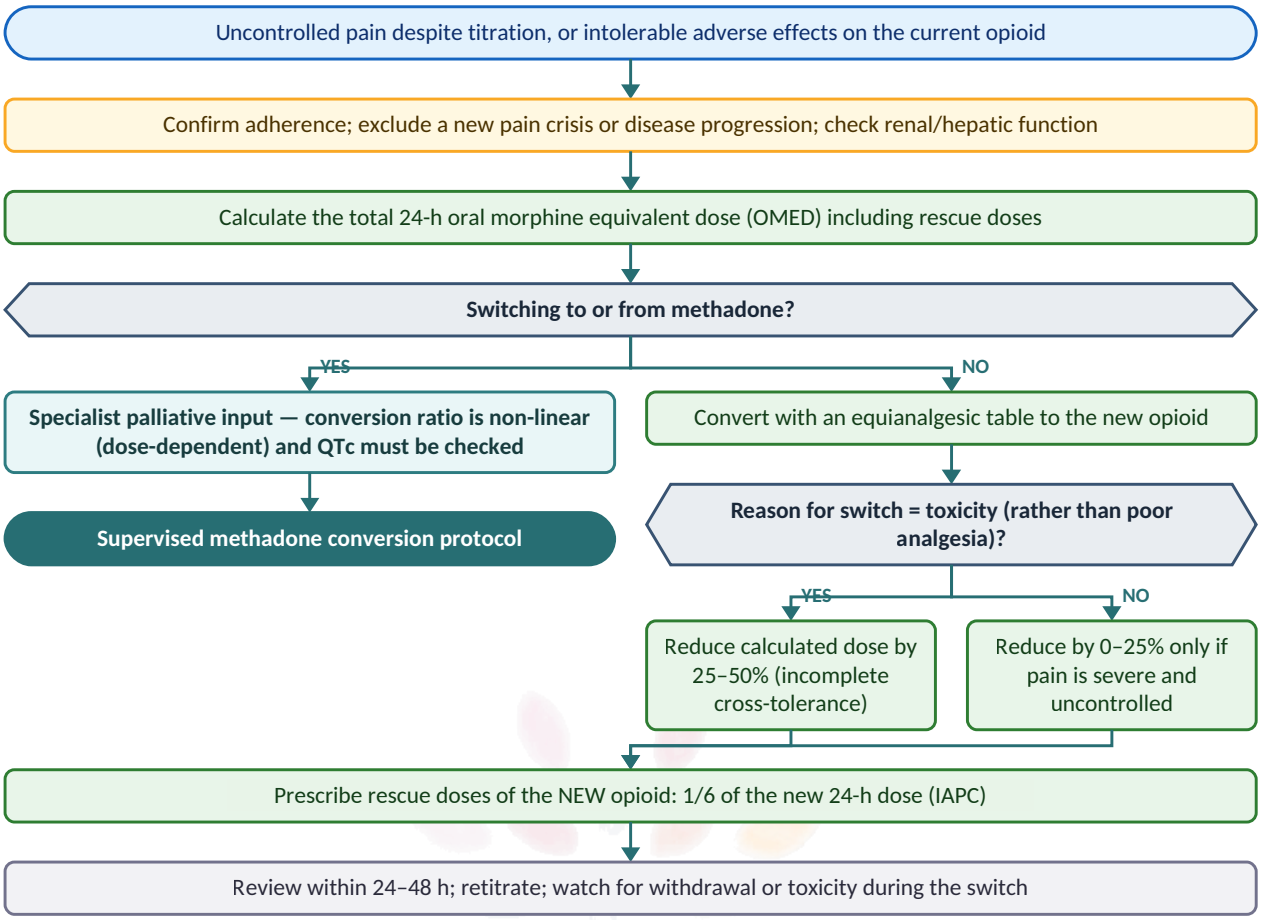
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ALGORITHM ATLAS · Algo 2

Algorithm: Opioid Rotation

Read the text ›

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Fentanyl patch: start new patch and stop the old opioid at the appropriate time (12 h overlap for SR morphine).
- Renal failure is the commonest reason to rotate from morphine — choose fentanyl, buprenorphine or alfentanil.

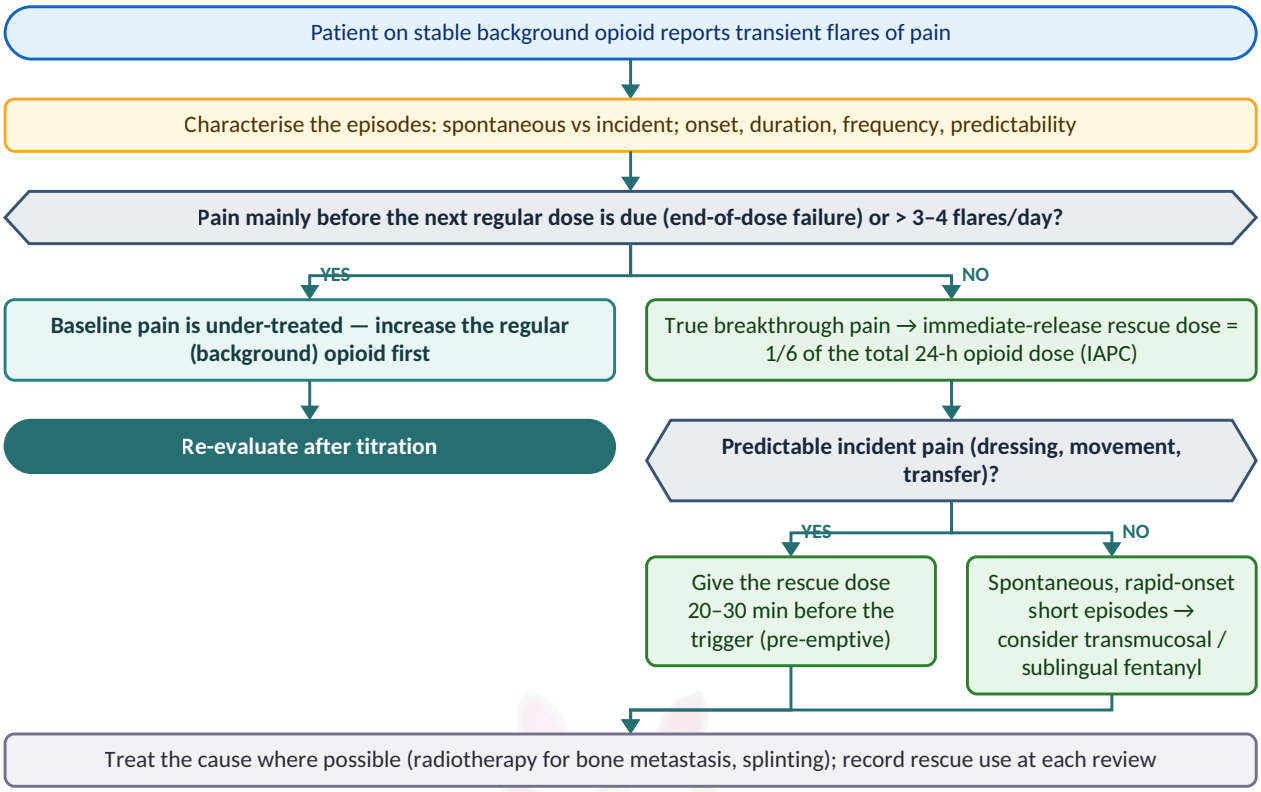
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ALGORITHM ATLAS · Algo 3

Algorithm: Approach to Breakthrough Pain

Read the text ›

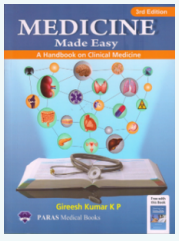
Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Typical breakthrough episode peaks in 3–5 min and lasts < 30 min — oral morphine (onset 20–30 min) is often too slow.
- Transmucosal fentanyl doses are titrated independently, not as a % of background dose.

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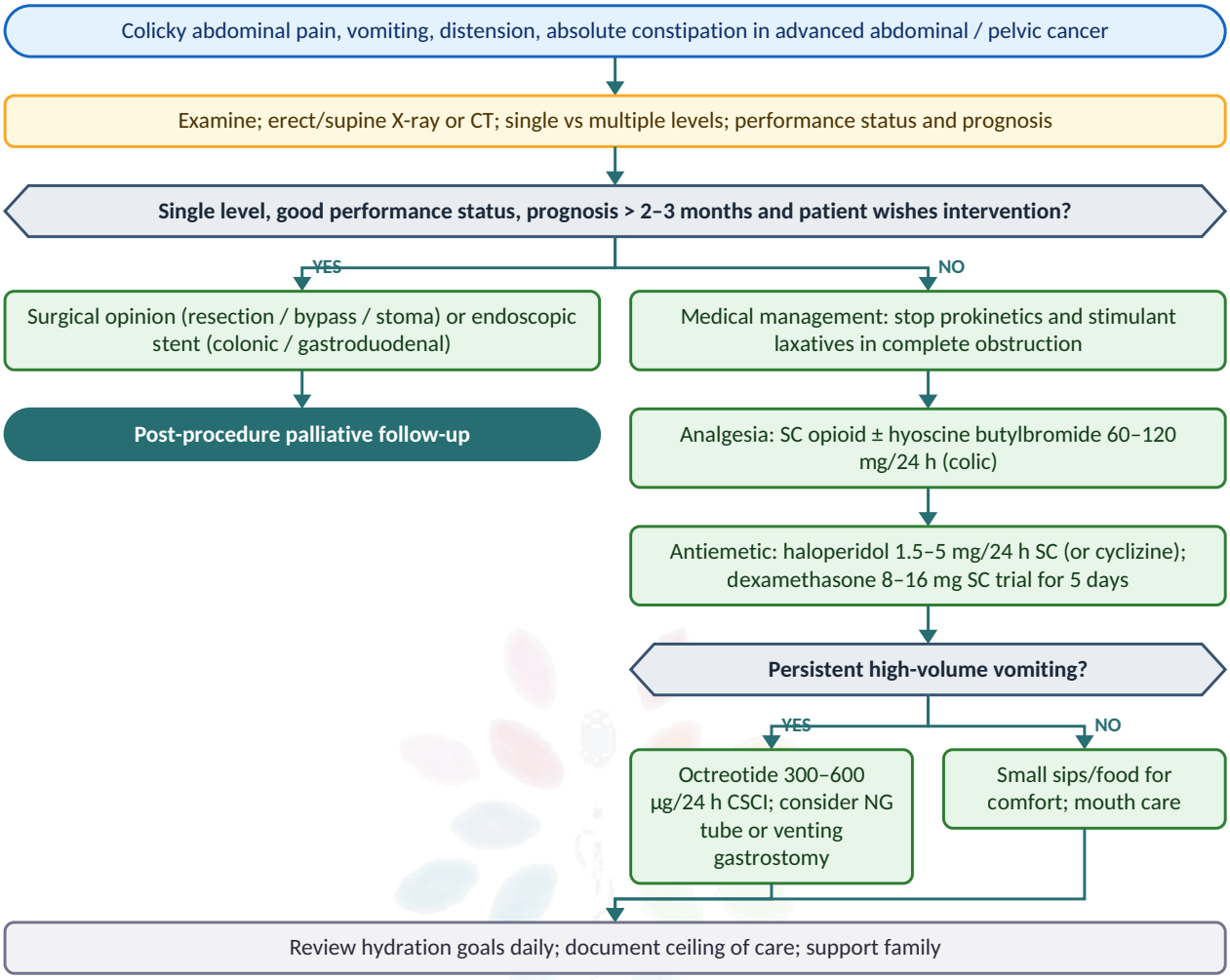
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ALGORITHM ATLAS · Algo 4

Algorithm: Approach to Malignant Bowel Obstruction

Read the text ›

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

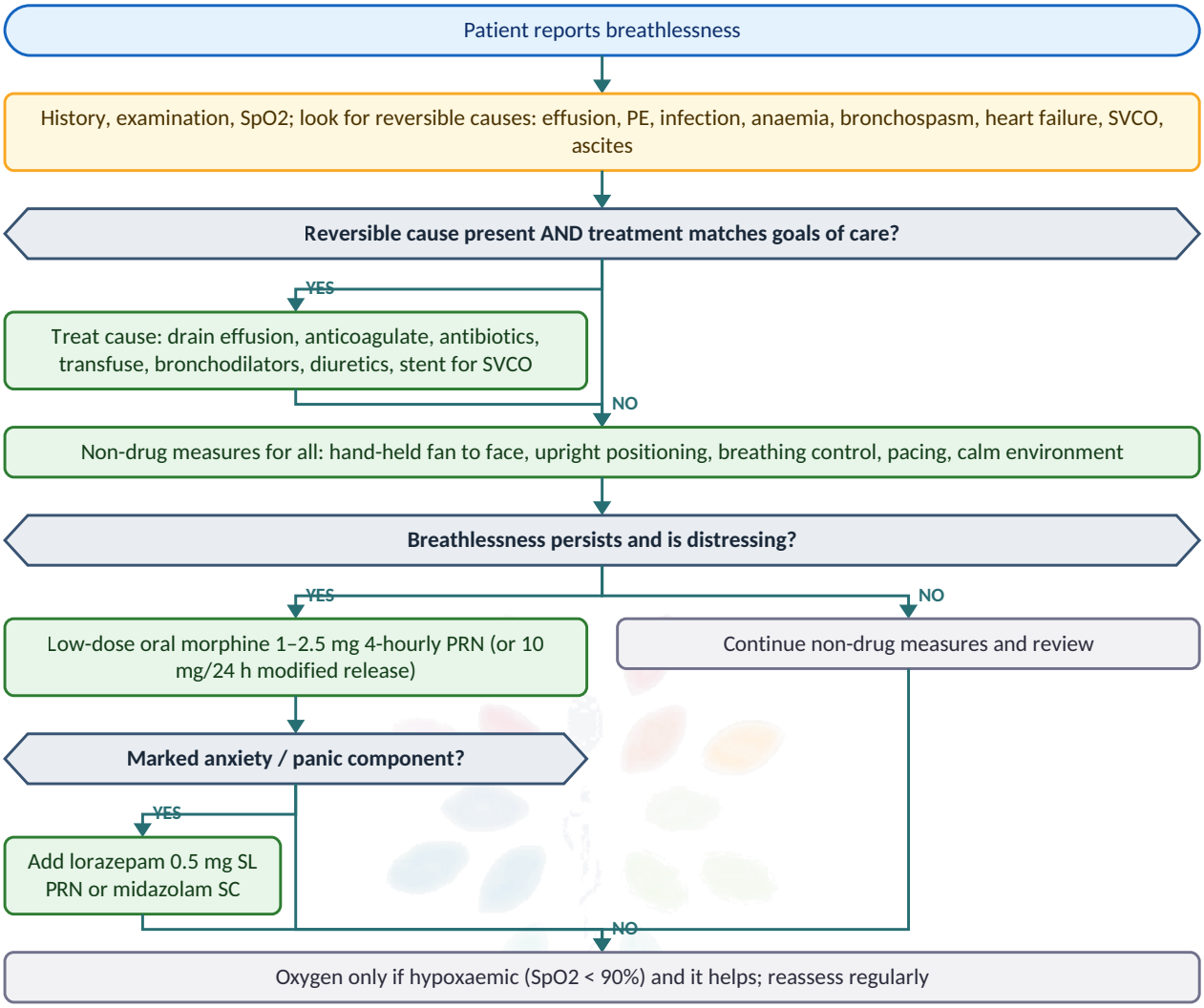
- Metoclopramide is used only in partial (functional) obstruction without colic.
- Venting PEG frees the patient from a nasogastric tube in refractory obstruction.

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ALGORITHM ATLAS · Algo 5

Algorithm: Approach to Breathlessness (Dyspnoea) in Palliative Care

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Opioids reduce the sensation of breathlessness without clinically significant respiratory depression at low doses.
- The fan directed at the face (trigeminal stimulation) is evidence-based and costless.

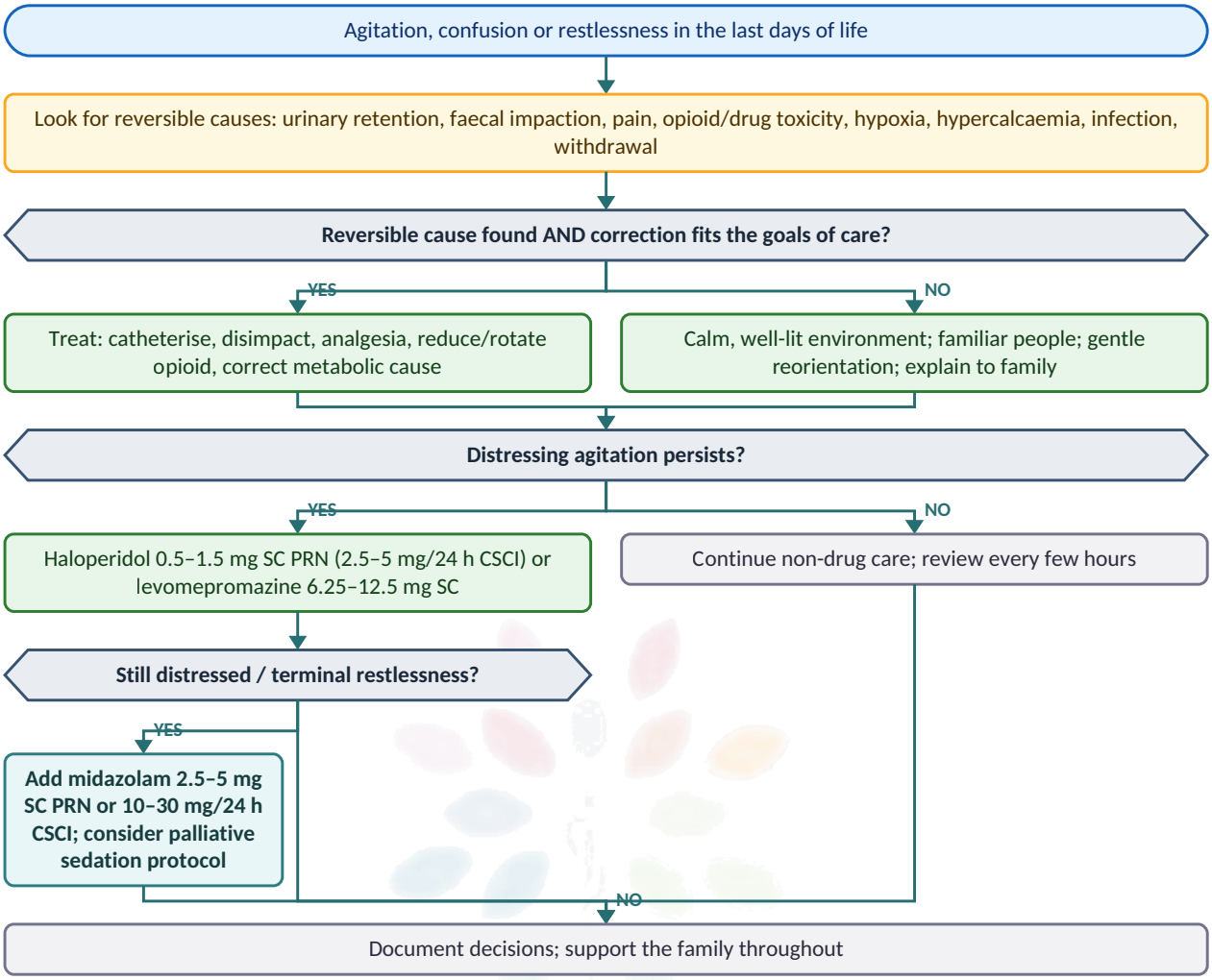
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ALGORITHM ATLAS · Algo 6

Algorithm: Approach to Terminal Delirium / Agitation

Read the text ›

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

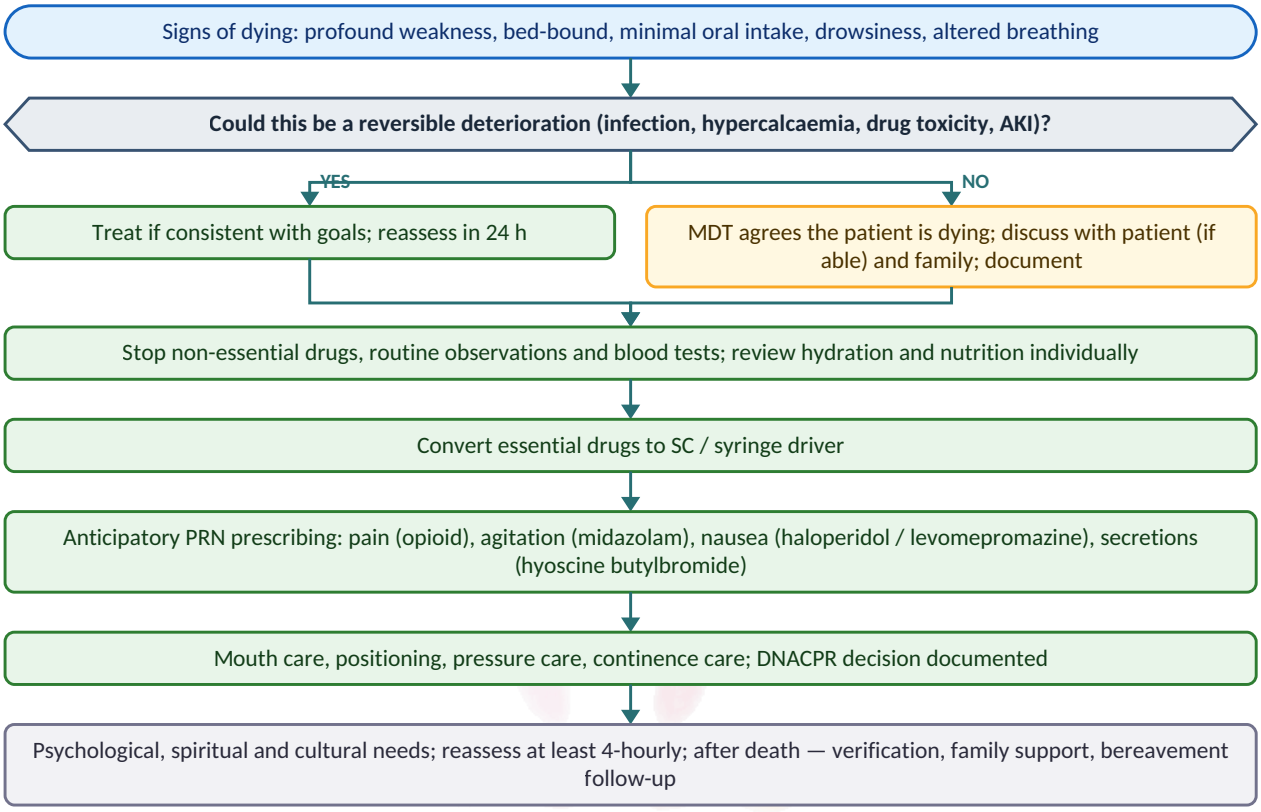
- Benzodiazepine alone can worsen delirium — give it with, not instead of, an antipsychotic (unless alcohol withdrawal).
- Hypoactive delirium is common and often missed.

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ALGORITHM ATLAS · Algo 7

Algorithm: Care in the Last Hours of Life (The Dying Phase)

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

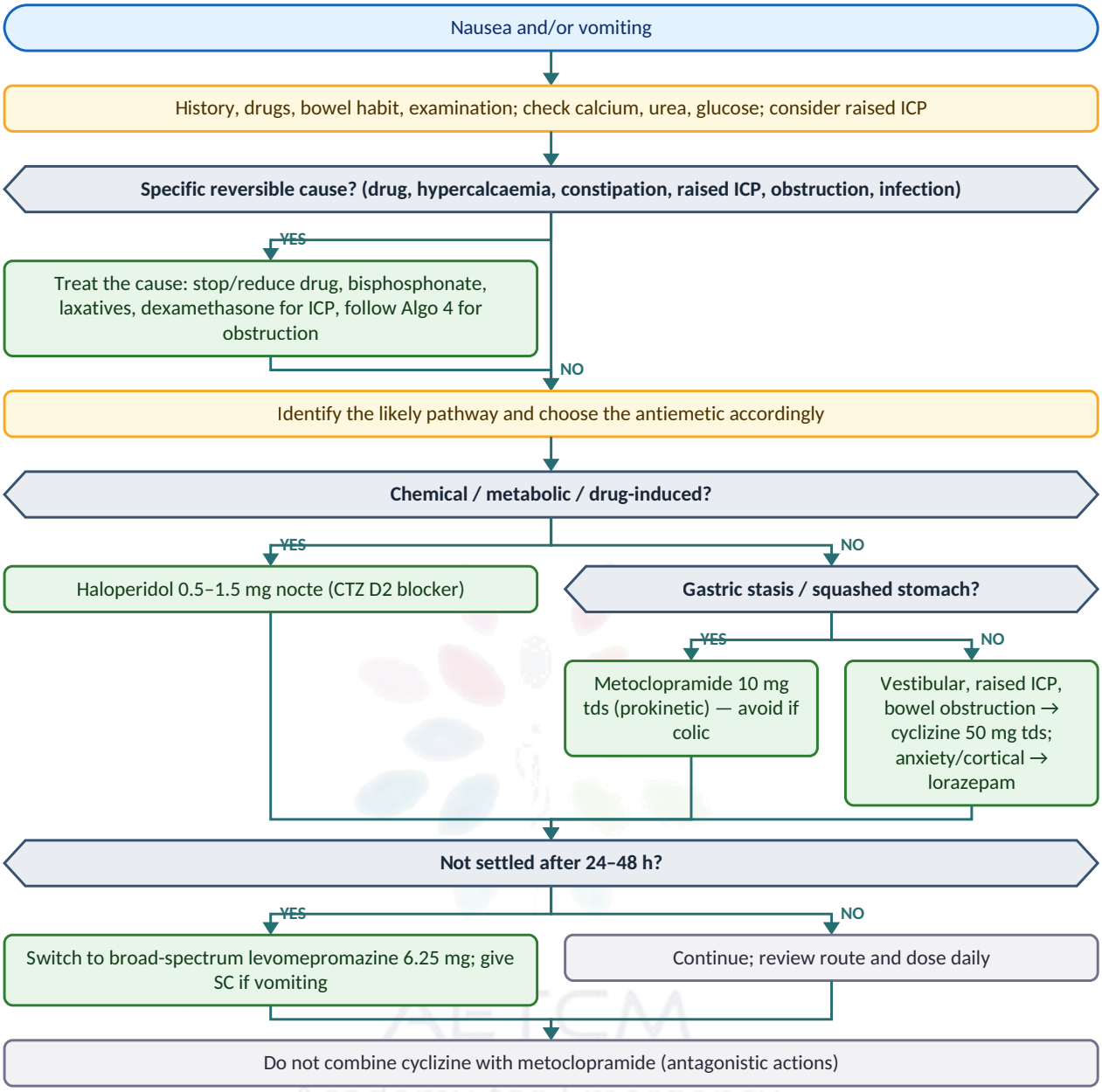
- Recognising dying is a team diagnosis — revisit it if the patient improves.
- Rituals (e.g. Ganga jal, prayers) matter to Indian families — ask, don't assume.

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ALGORITHM ATLAS · Algo 8

Algorithm: Approach to Nausea and Vomiting in Palliative Care

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

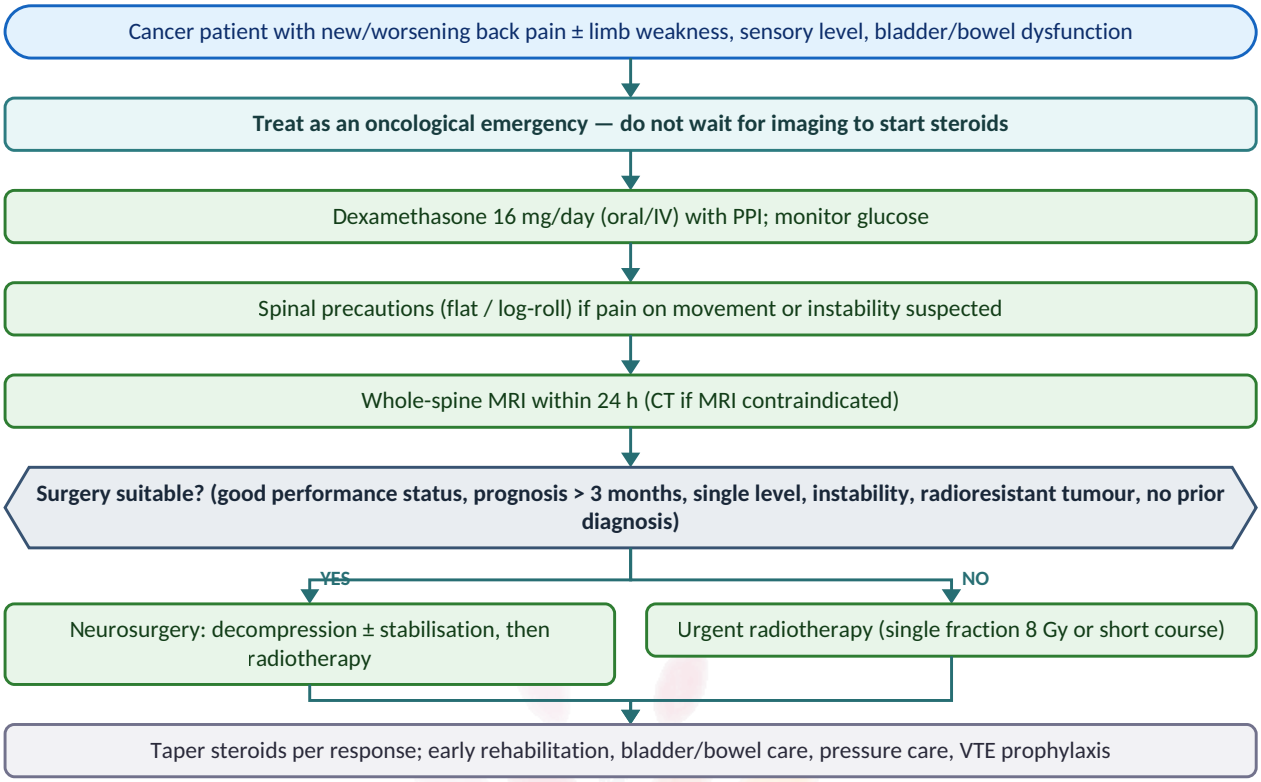
- Choose antiemetic by mechanism, not by habit.
- Ondansetron is mainly for chemo-/radiotherapy-induced emesis and constipates.

ALGORITHM ATLAS · Algo 9

Algorithm: Approach to Suspected Malignant Spinal Cord Compression

Read the text ›

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Ambulatory status at diagnosis is the strongest predictor of outcome.
- Patchell trial (2005): surgery + RT kept more patients walking than RT alone in selected cases.

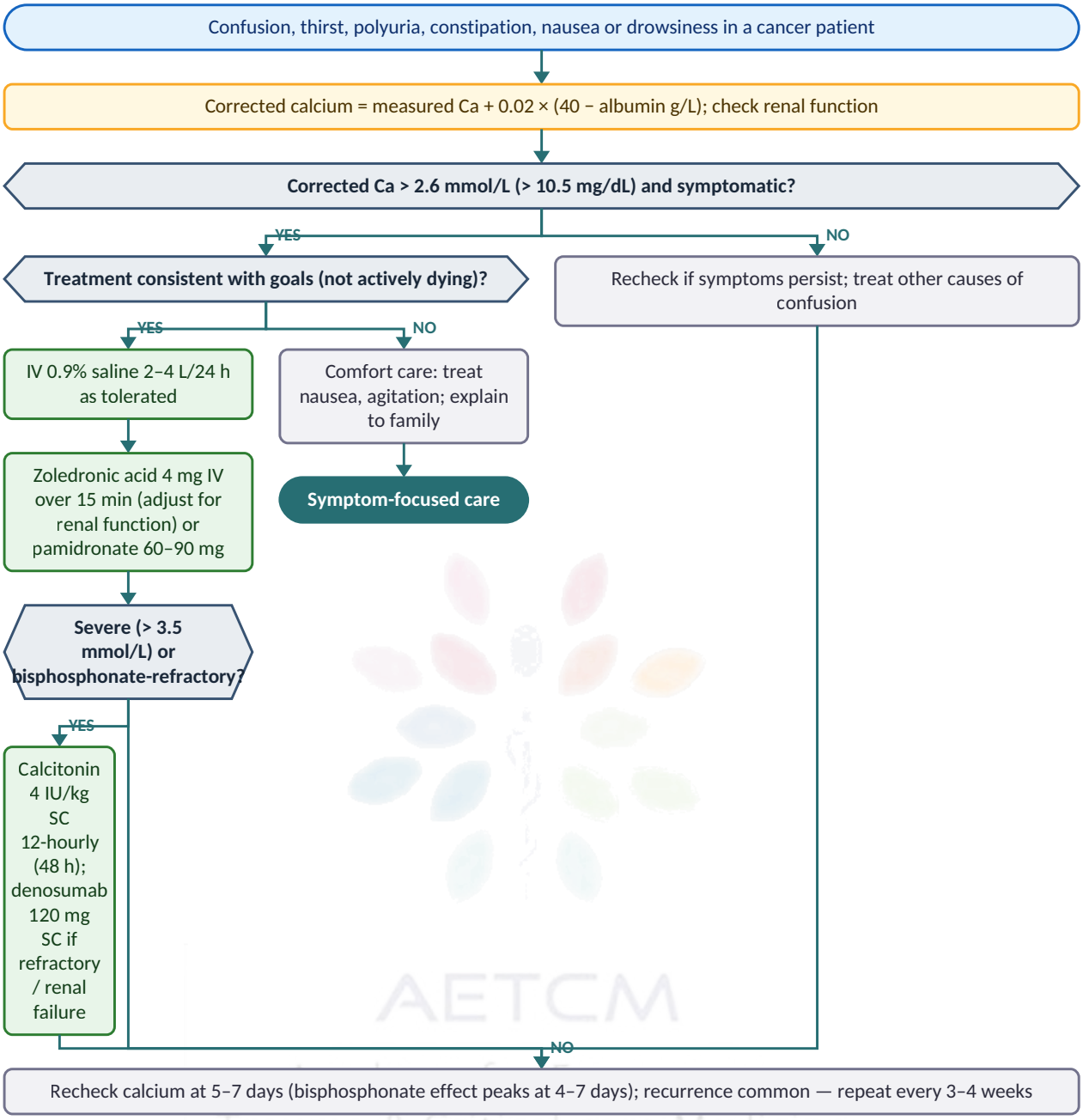
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ALGORITHM ATLAS · Algo 10

Algorithm: Approach to Hypercalcaemia of Malignancy

Read the text ›

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

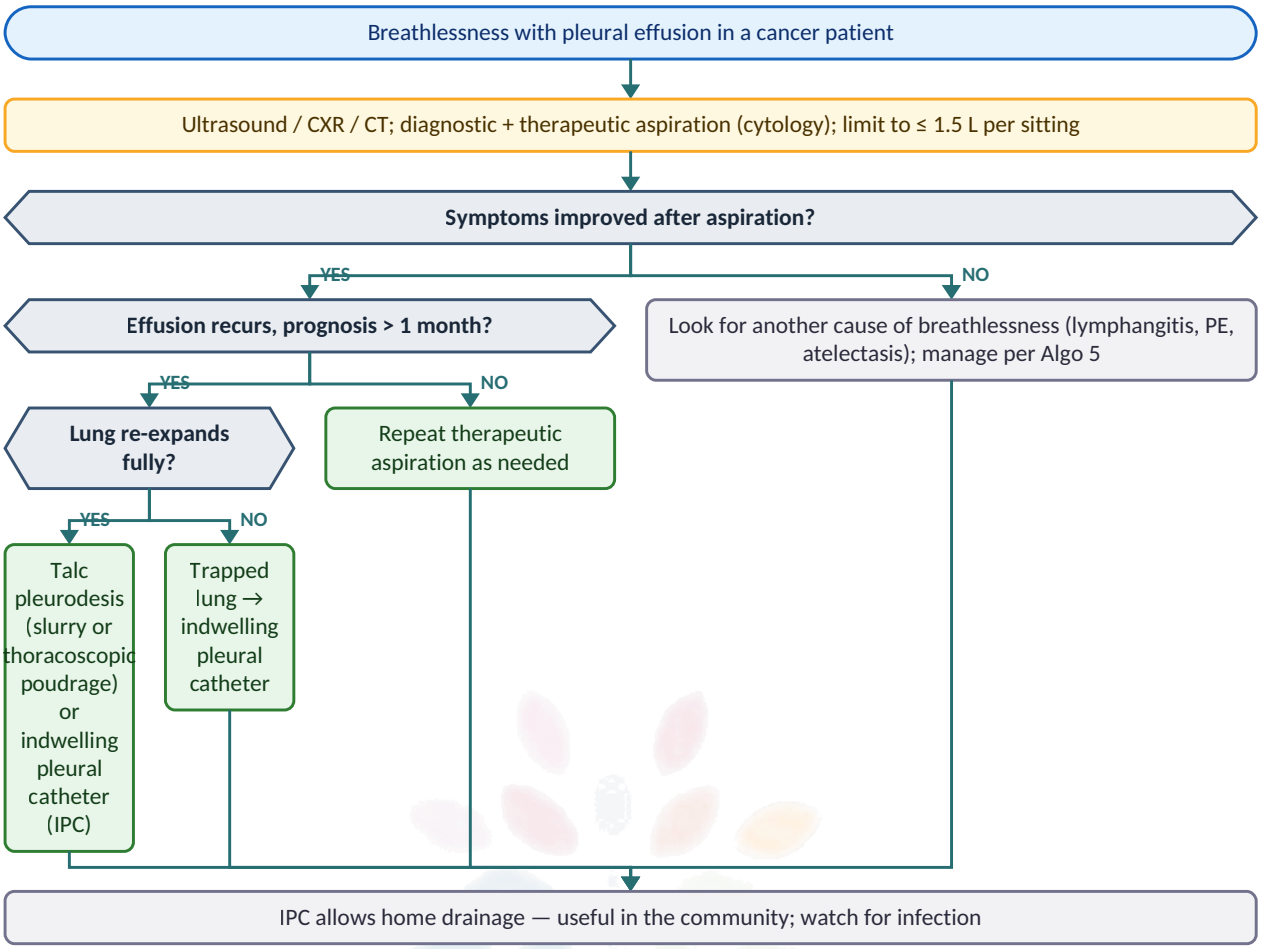
- Median survival after cancer hypercalcaemia is often weeks to months — discuss goals.
- PTHrP-mediated humoral hypercalcaemia is commonest (squamous carcinomas).

ALGORITHM ATLAS · Algo 11

Algorithm: Approach to Malignant Pleural Effusion

Read the text ›

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

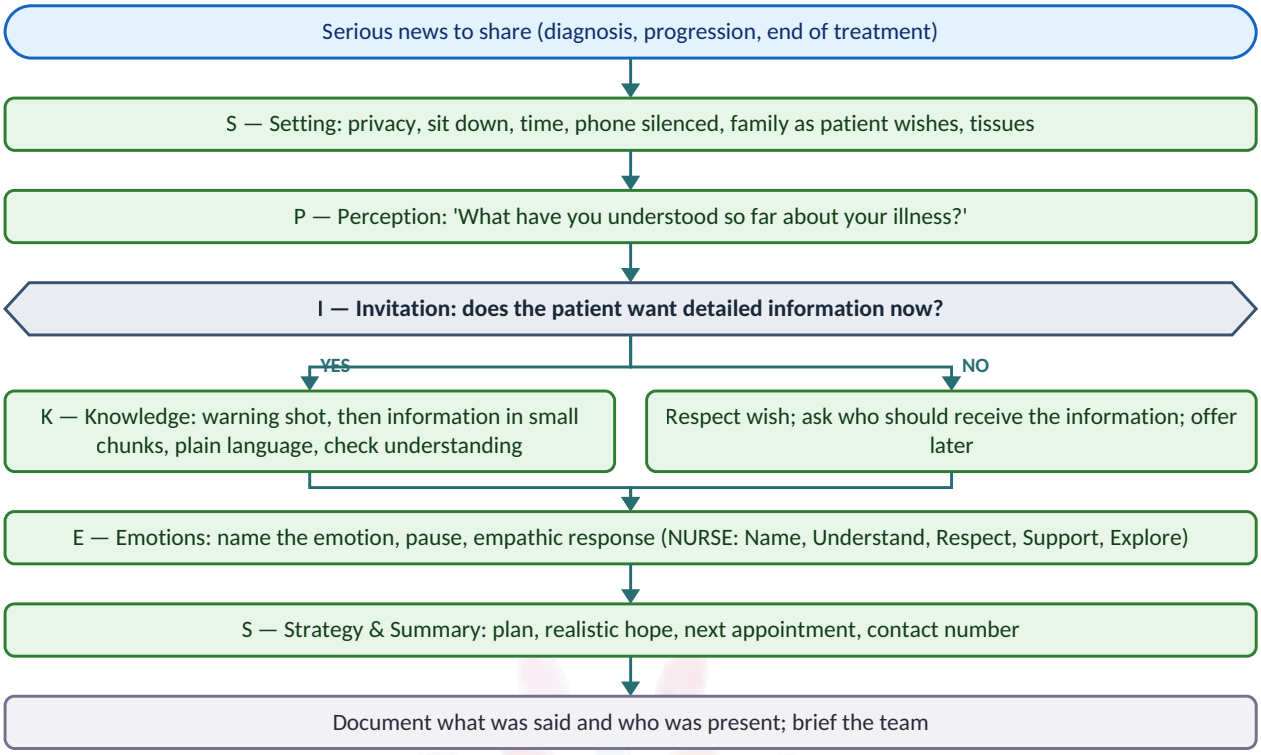
- Re-expansion pulmonary oedema: stop drainage if chest tightness or cough develops.
- AMPLE-2 and TIME2 trials support IPC as first-line in many patients.

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ALGORITHM ATLAS · Algo 12

Algorithm: Breaking Bad News — the SPIKES Protocol

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Silence is a skill — allow it after the news.
- In India, families often request non-disclosure — explore their fears and the patient's wishes, don't simply comply.

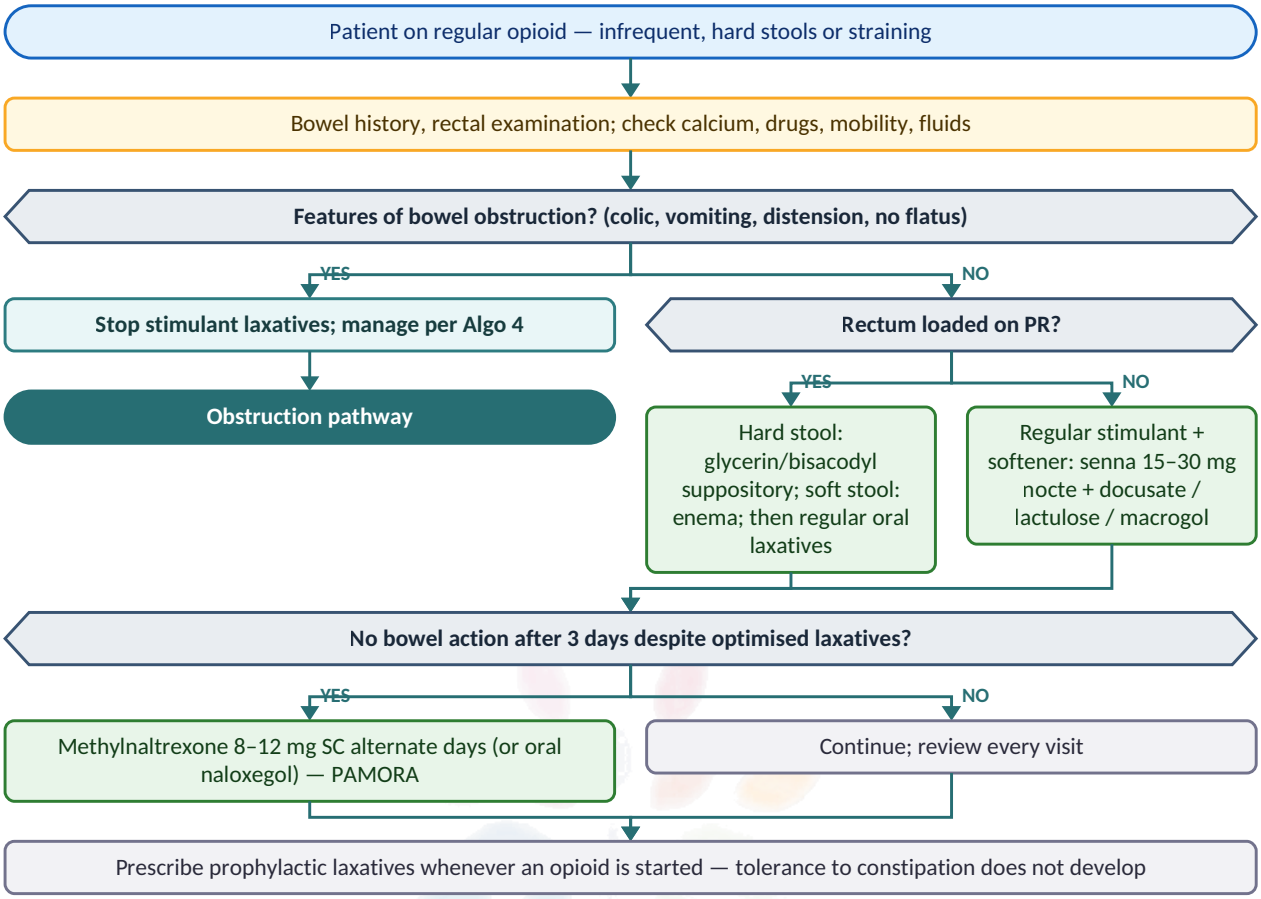
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ALGORITHM ATLAS · Algo 13

Algorithm: Approach to Opioid-Induced Constipation

Read the text ›

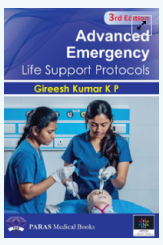
Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- PAMORAs are contraindicated in known or suspected obstruction.
- Lactulose causes bloating; macrogol is often better tolerated.

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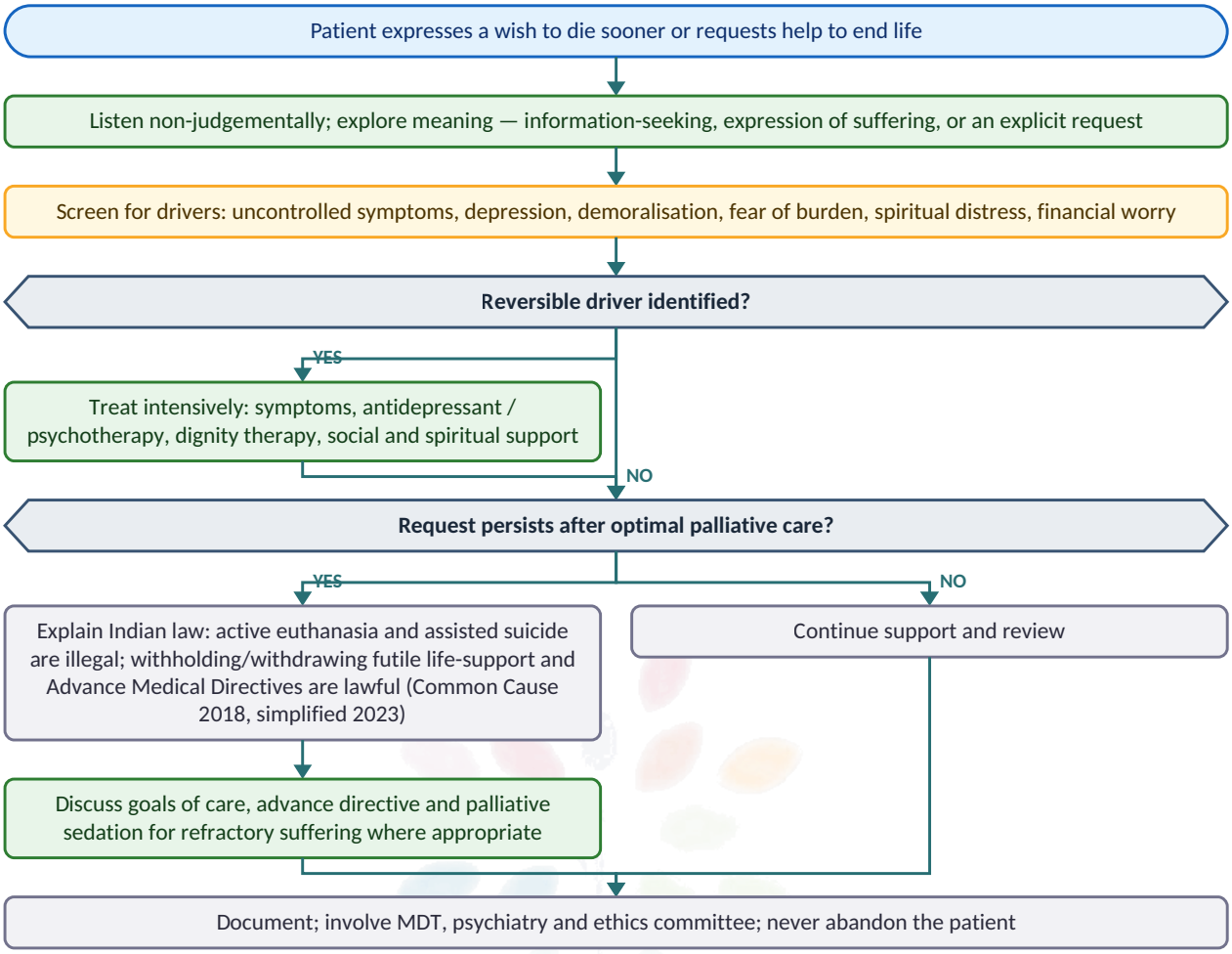
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ALGORITHM ATLAS · Algo 14

Algorithm: Approach to a Request for Hastened Death

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Asking about a wish to die does not increase risk — it opens the door to care.
- Demoralisation (hopelessness, loss of meaning) is distinct from depression and responds to meaning-centred therapy.

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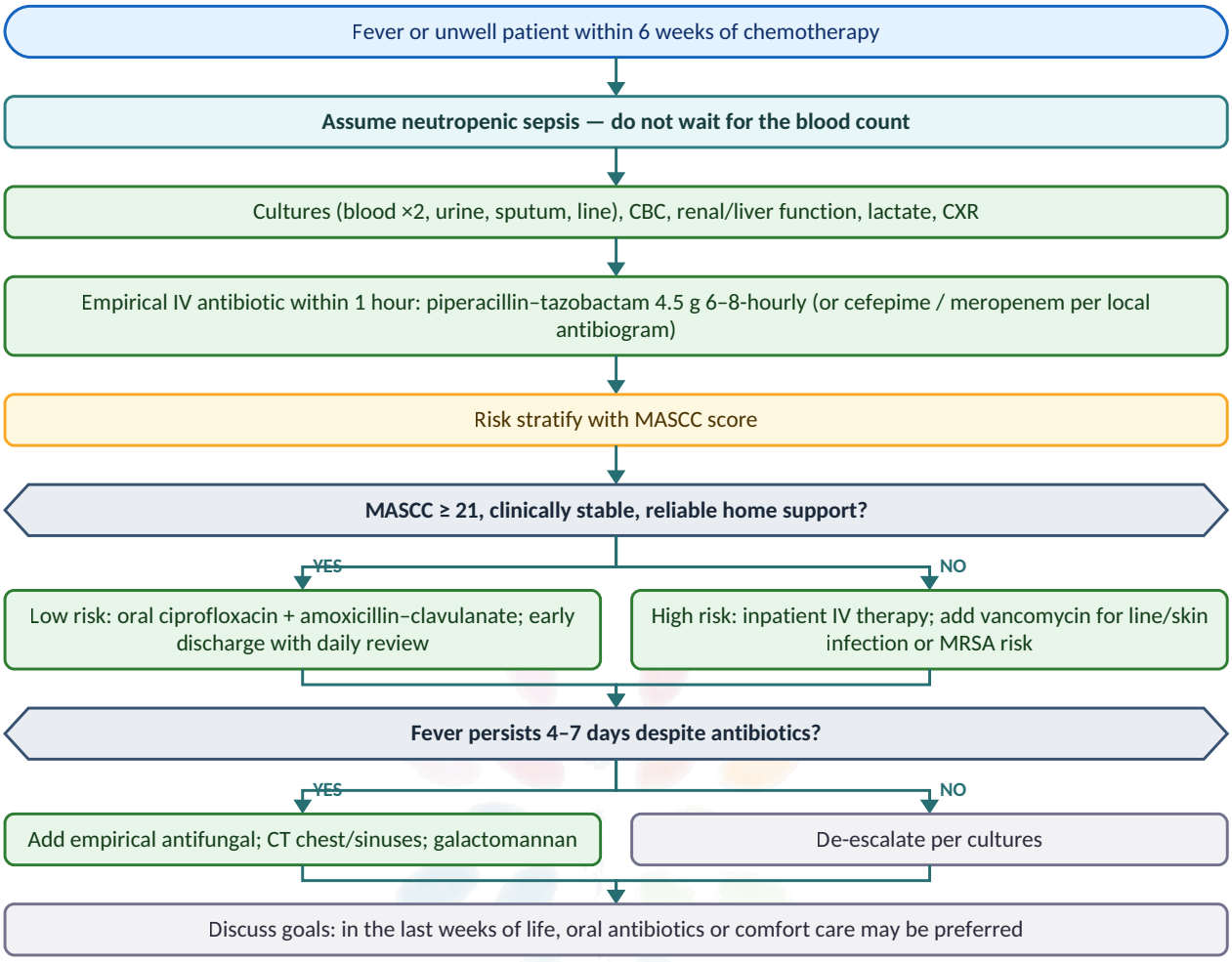
ALGORITHM ATLAS · Algo 15

Algorithm: Febrile Neutropenia

Read the text ›

Temperature ≥ 38.3 °C once (or ≥ 38.0 °C for 1 h) with ANC < 500/µL after chemotherapy.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Time to first antibiotic dose is the key quality indicator.
- G-CSF is not routinely given for established febrile neutropenia.

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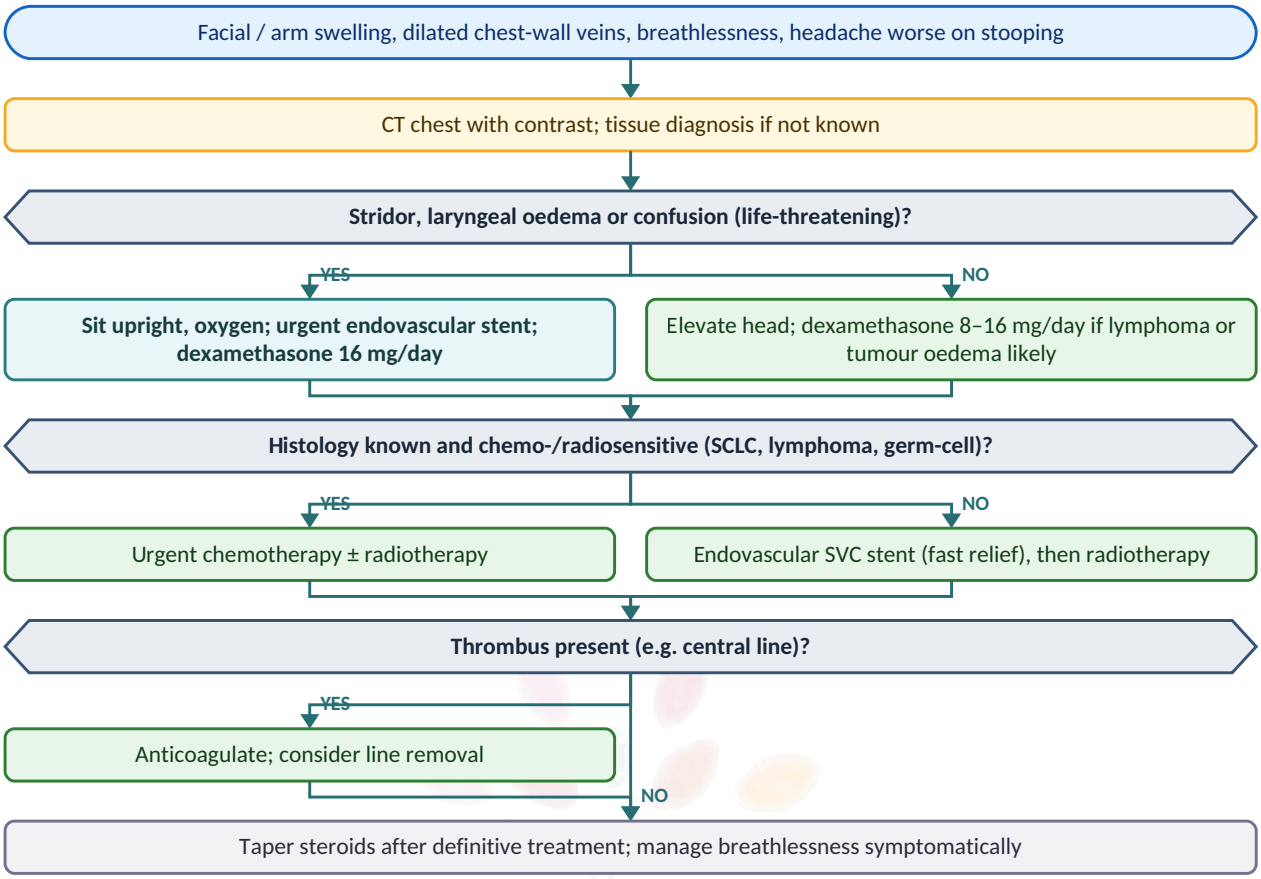
ALGORITHM ATLAS · Algo 16

Algorithm: Superior Vena Cava Obstruction

Read the text ›

Obstruction of SVC flow by tumour (lung cancer, lymphoma) or thrombus.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Obtain histology before steroids if lymphoma is suspected — steroids can mask the diagnosis.
- Stenting gives relief within 24–72 h in > 90%.

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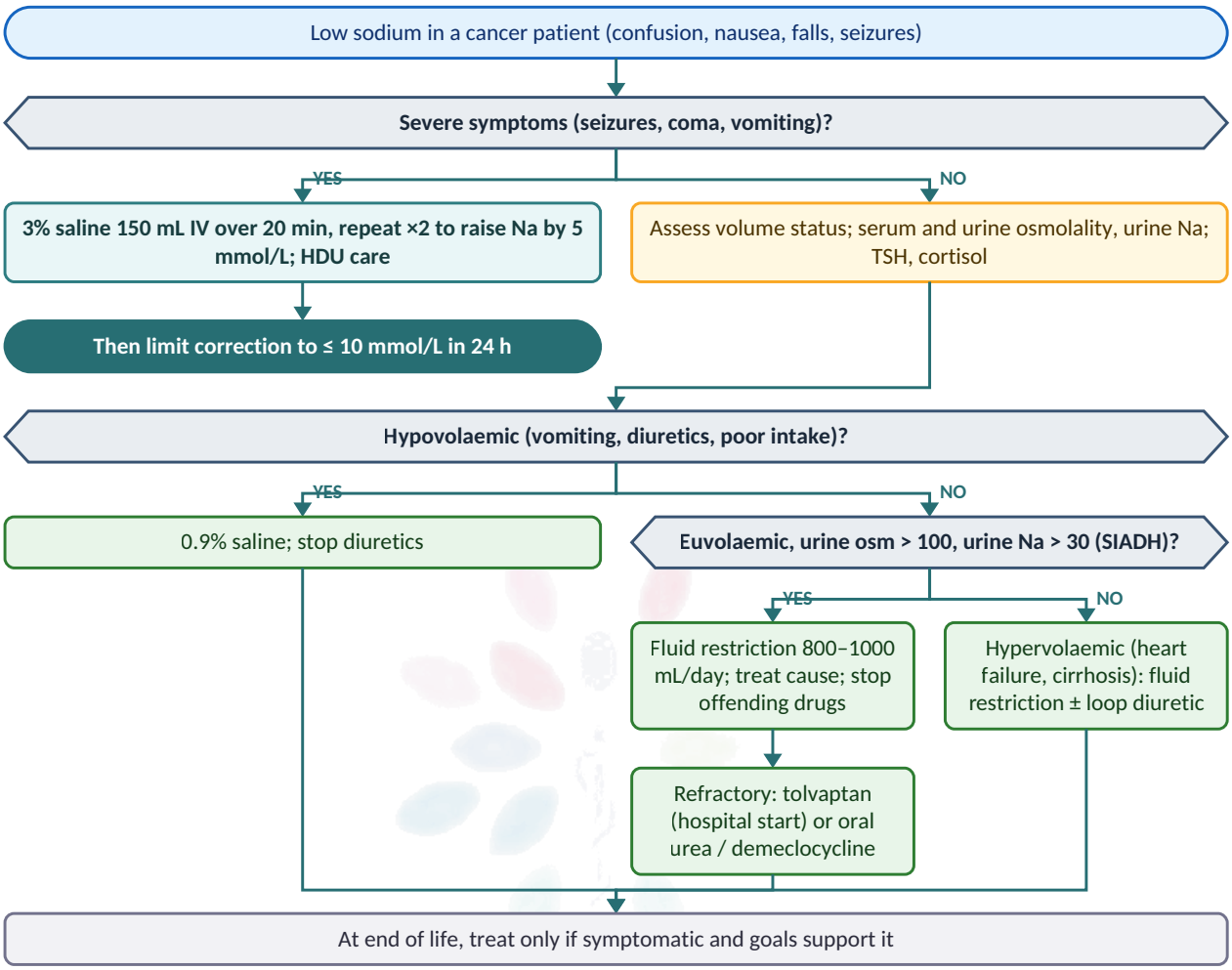
ALGORITHM ATLAS · Algo 17

Algorithm: Hyponatraemia and SIADH in Malignancy

Read the text ›

Na < 135 mmol/L; SIADH is common with small-cell lung cancer and drugs (carbamazepine, SSRIs, cyclophosphamide).

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Over-rapid correction risks osmotic demyelination — check Na 4–6-hourly initially.
- Fluid restriction is often burdensome in the dying — individualise.

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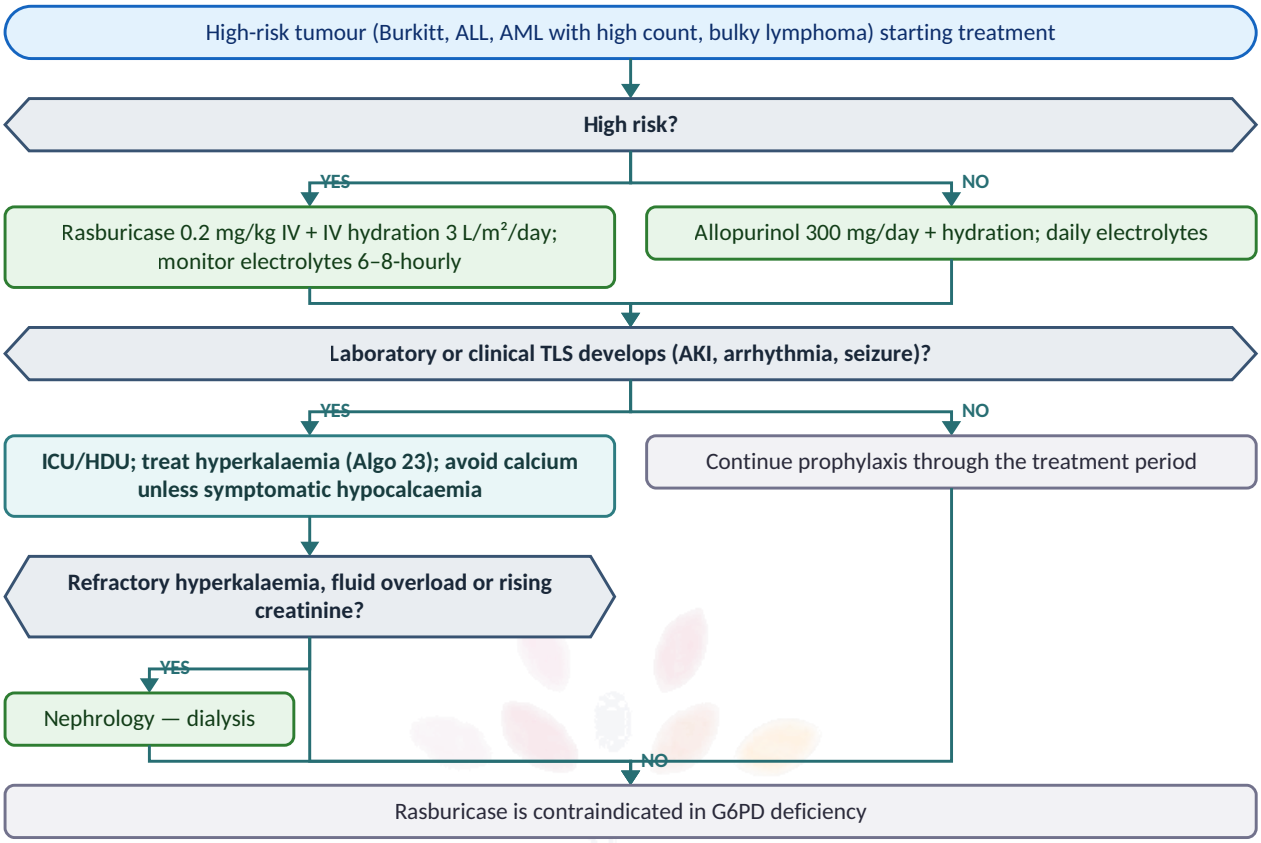
ALGORITHM ATLAS · Algo 18

Algorithm: Tumour Lysis Syndrome

Read the text ›

Hyperuricaemia, hyperkalaemia, hyperphosphataemia and hypocalcaemia after cytotoxic therapy of bulky, chemosensitive tumours.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Cairo–Bishop criteria define laboratory and clinical TLS.
- Do not alkalinise urine routinely — calcium phosphate may precipitate.

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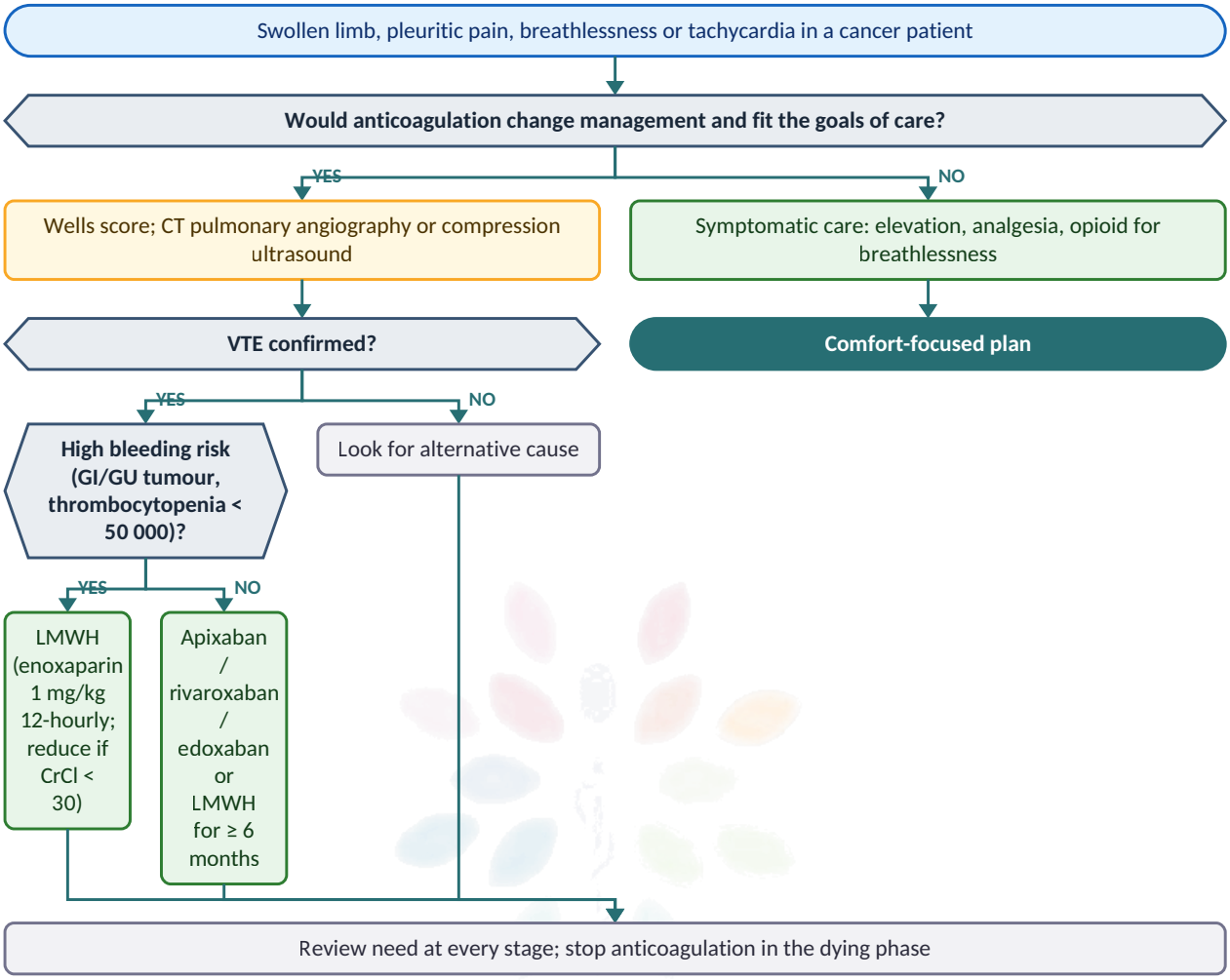
ALGORITHM ATLAS · Algo 19

Algorithm: Suspected Venous Thromboembolism in Cancer

Read the text ›

Cancer raises VTE risk four- to seven-fold; decisions must weigh bleeding risk and prognosis.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- CLOT trial: LMWH superior to warfarin in cancer VTE.
- Platelets < 50 000: halve dose LMWH or withhold.

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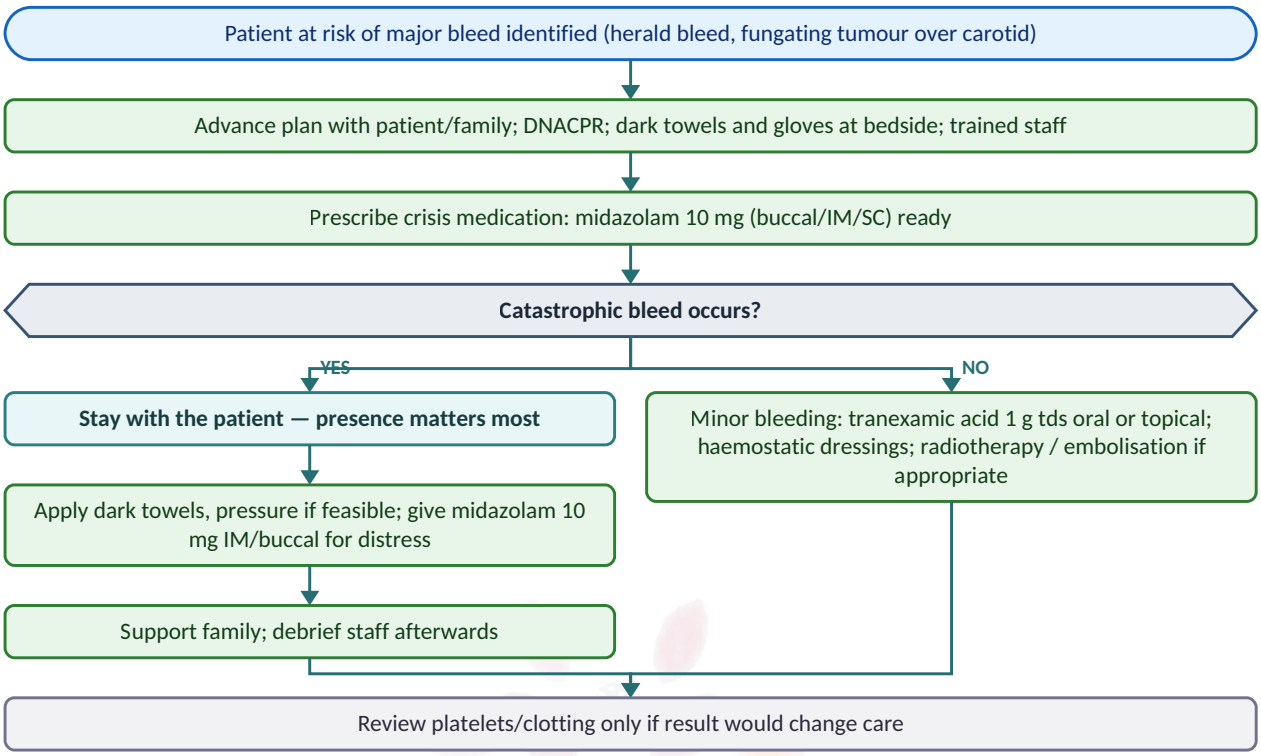
ALGORITHM ATLAS · Algo 20

Algorithm: Catastrophic Haemorrhage at the End of Life

Read the text ›

Anticipate in head-and-neck cancers near major vessels, haemoptysis, and GI tumours.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Death from catastrophic bleed is usually within minutes — sedation may not take effect; calm presence is the key intervention.
- Use IM route if no IV access — SC absorption is unreliable in shock.

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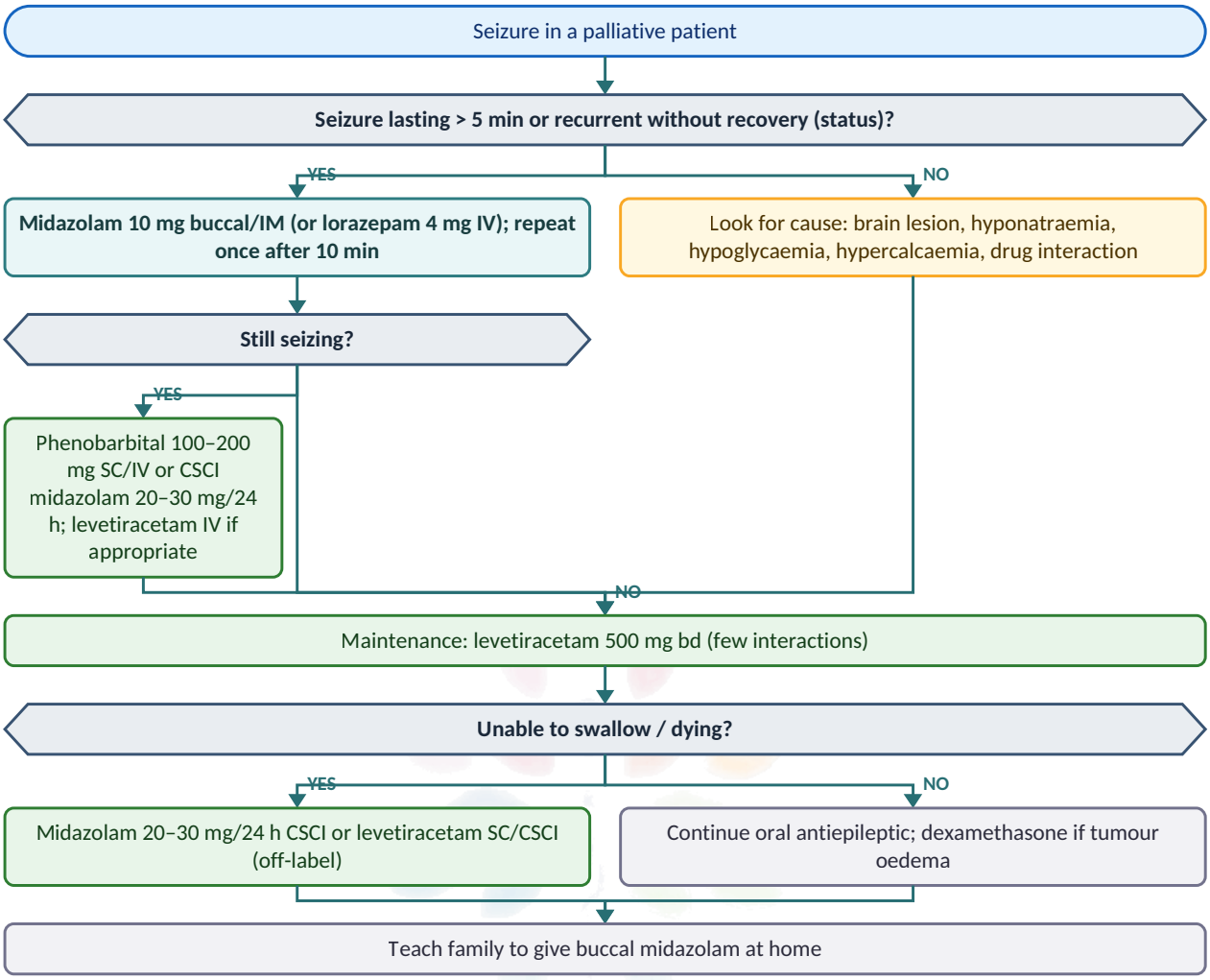
ALGORITHM ATLAS · Algo 21

Algorithm: Seizures in Palliative Care

Read the text ›

Common with primary brain tumours and metastases; also metabolic causes.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Avoid enzyme-inducing antiepileptics (phenytoin, carbamazepine) with chemotherapy and dexamethasone.
- No primary seizure prophylaxis for brain metastases without seizures.

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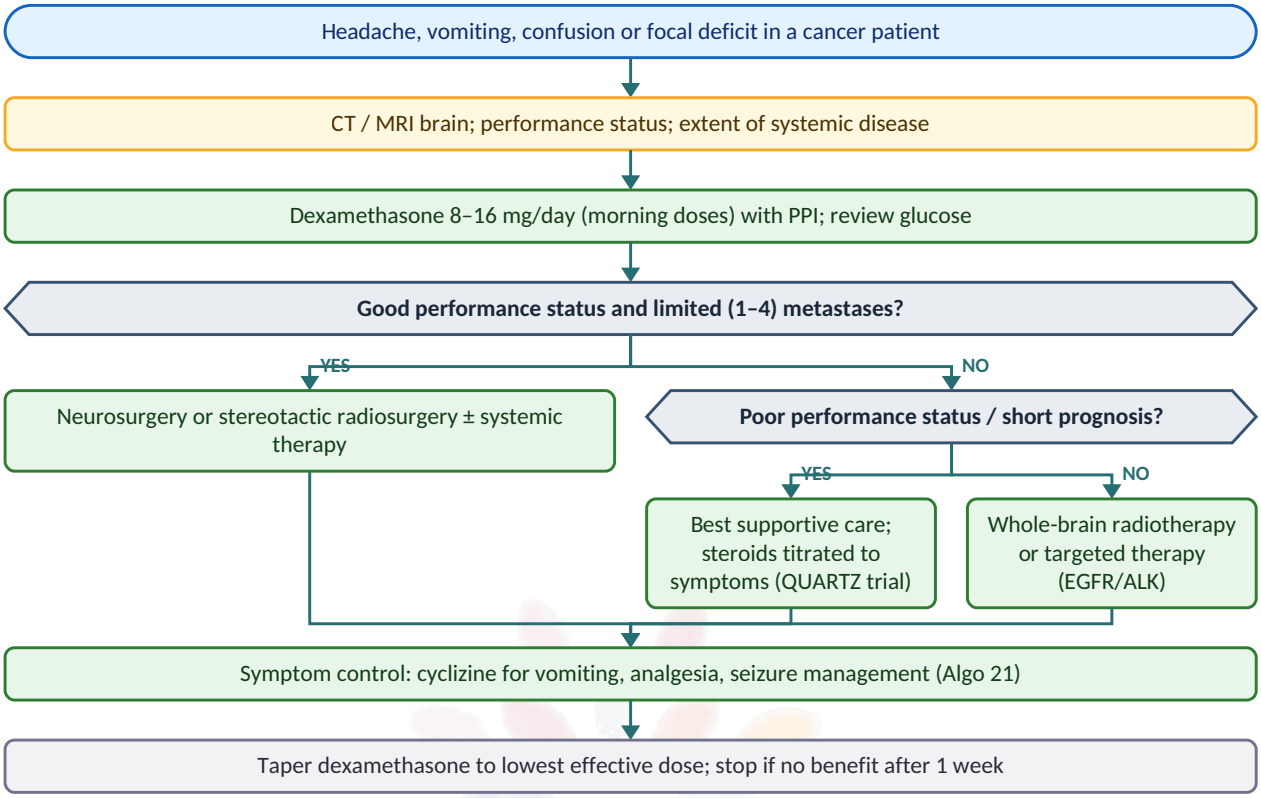
ALGORITHM ATLAS · Algo 22

Algorithm: Raised Intracranial Pressure and Brain Metastases

Read the text ›

Headache worse in the morning, vomiting, drowsiness, papilloedema, focal deficits.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- QUARTZ: WBRT added little to quality-adjusted survival in poor-prognosis NSCLC brain metastases.
- Give steroids before 2 pm to reduce insomnia.

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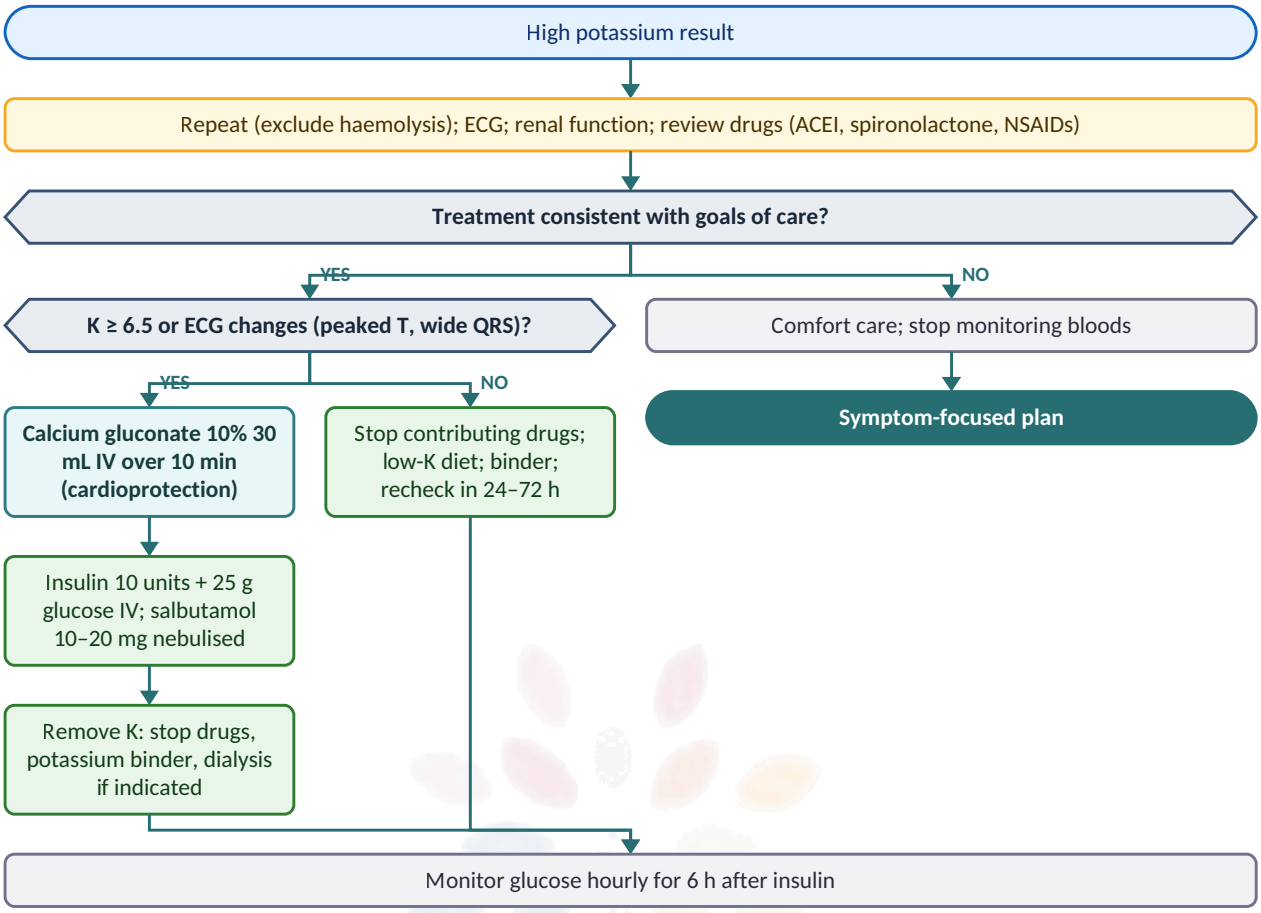
ALGORITHM ATLAS · Algo 23

Algorithm: Hyperkalaemia

Read the text ›

K > 5.5 mmol/L; urgent if ≥ 6.5 mmol/L or ECG changes.

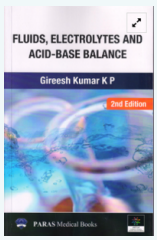
Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Calcium does not lower potassium — it stabilises the myocardium.
- In the dying, hyperkalaemia need not be treated.

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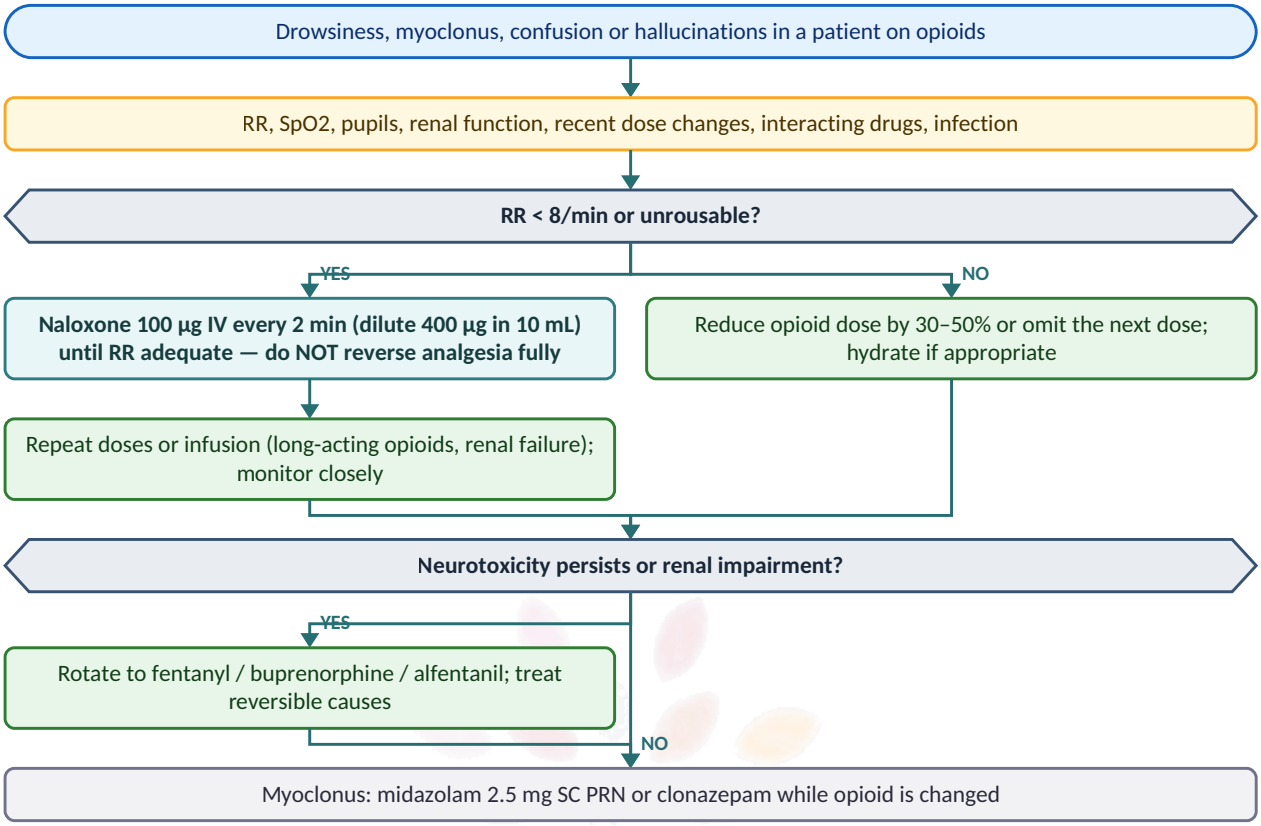
ALGORITHM ATLAS · Algo 24

Algorithm: Opioid Toxicity and Opioid-Induced Neurotoxicity

Read the text ›

Drowsiness, pinpoint pupils, myoclonus, hallucinations, hyperalgesia; often precipitated by renal impairment.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Full-dose naloxone in opioid-tolerant patients causes severe pain and withdrawal.
- Morphine-3- and -6-glucuronide accumulate in renal failure.

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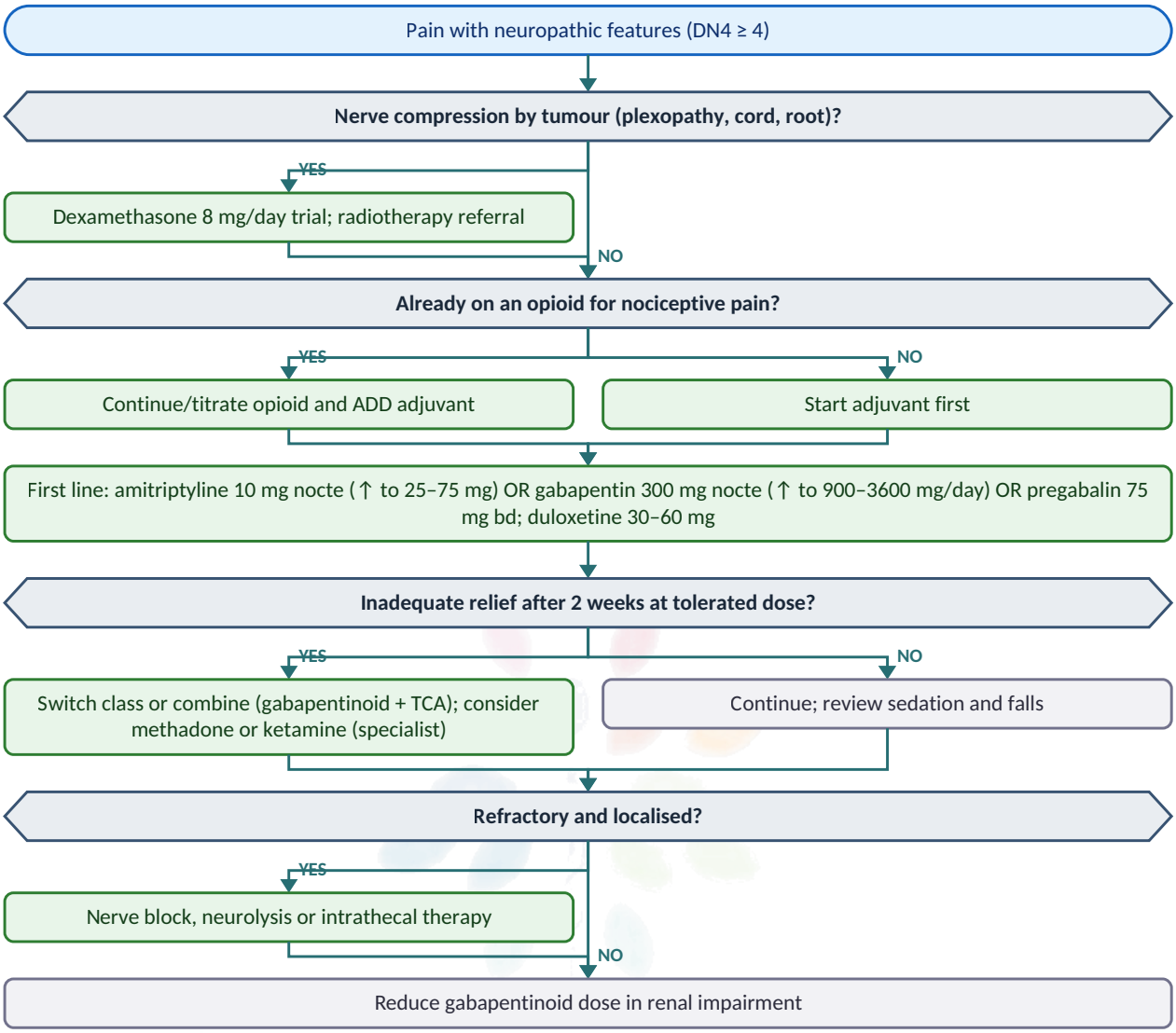
ALGORITHM ATLAS · Algo 25

Algorithm: Neuropathic Pain Pharmacotherapy

Read the text ›

Burning, shooting or electric pain, allodynia, in a neurological distribution.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Number needed to treat ~3–4 for TCAs; ~6–8 for gabapentinoids.
- Amitriptyline: caution in cardiac disease and elderly.

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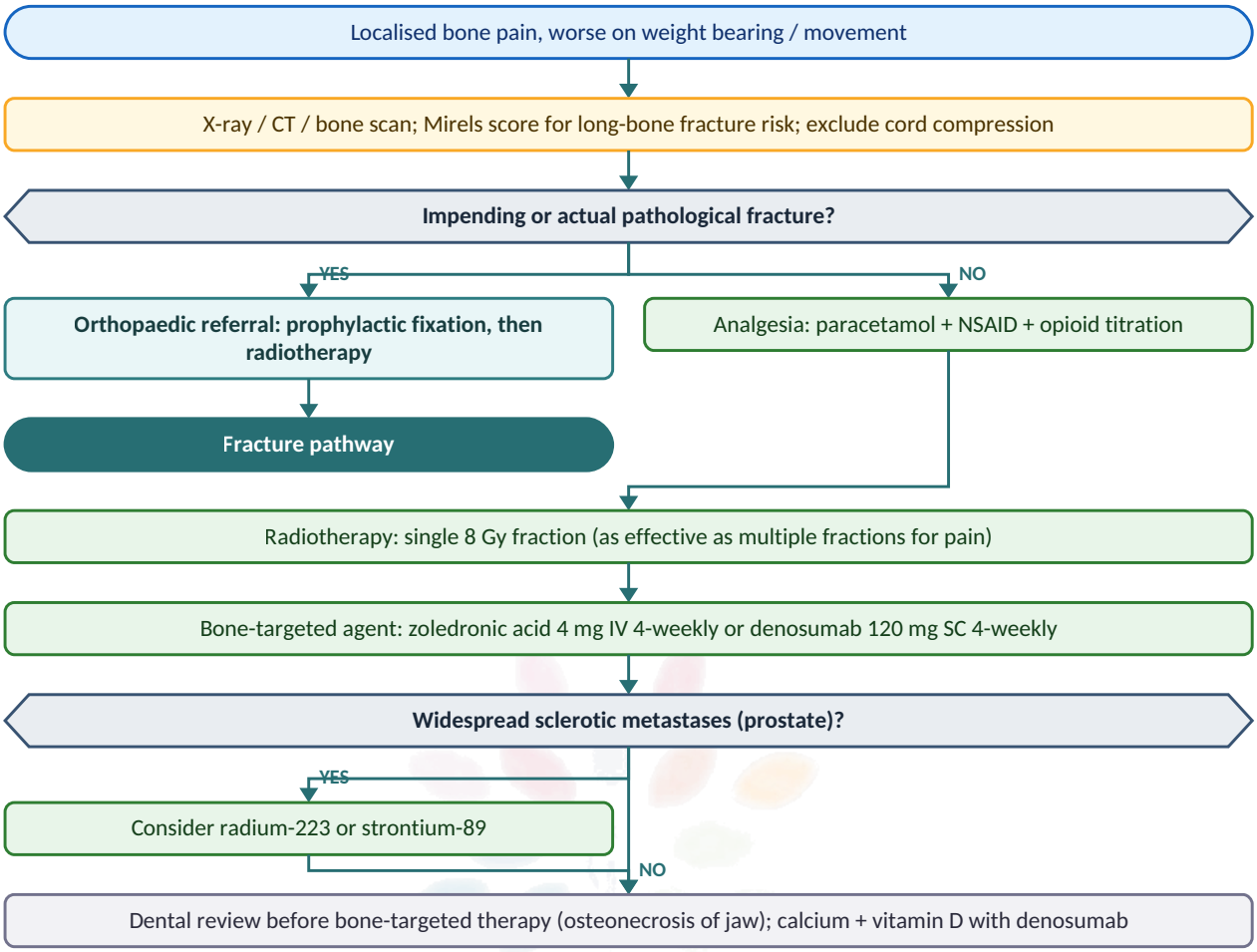
ALGORITHM ATLAS · Algo 26

Algorithm: Pain from Bone Metastases

Read the text ›

The commonest cause of cancer pain; risk of fracture and cord compression.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Pain relief after radiotherapy takes 2–4 weeks; pain flare in the first days — cover with dexamethasone.
- Mirels score ≥ 9 → prophylactic fixation.

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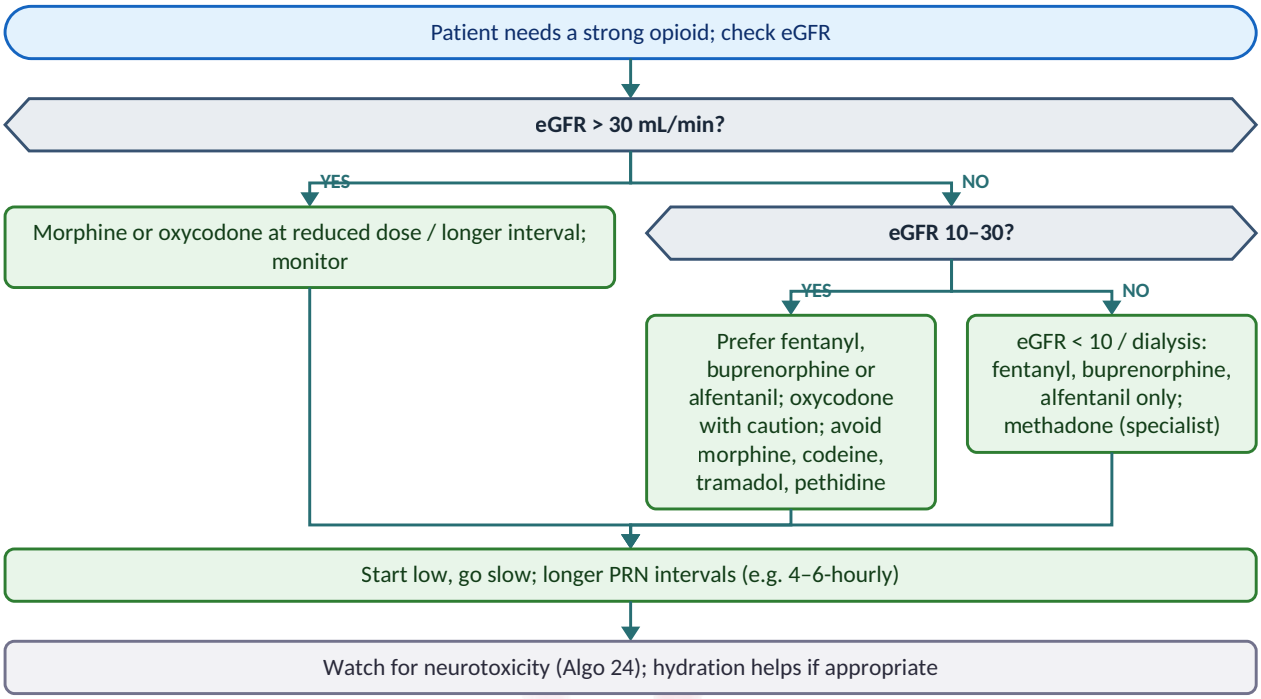
ALGORITHM ATLAS · Algo 27

Algorithm: Choosing an Opioid in Renal Impairment

Read the text ›

Active metabolites of morphine, codeine, tramadol and oxycodone accumulate when eGFR falls.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Fentanyl and alfentanil are hepatically metabolised to inactive metabolites.
- Hydromorphone is intermediate — use with caution.

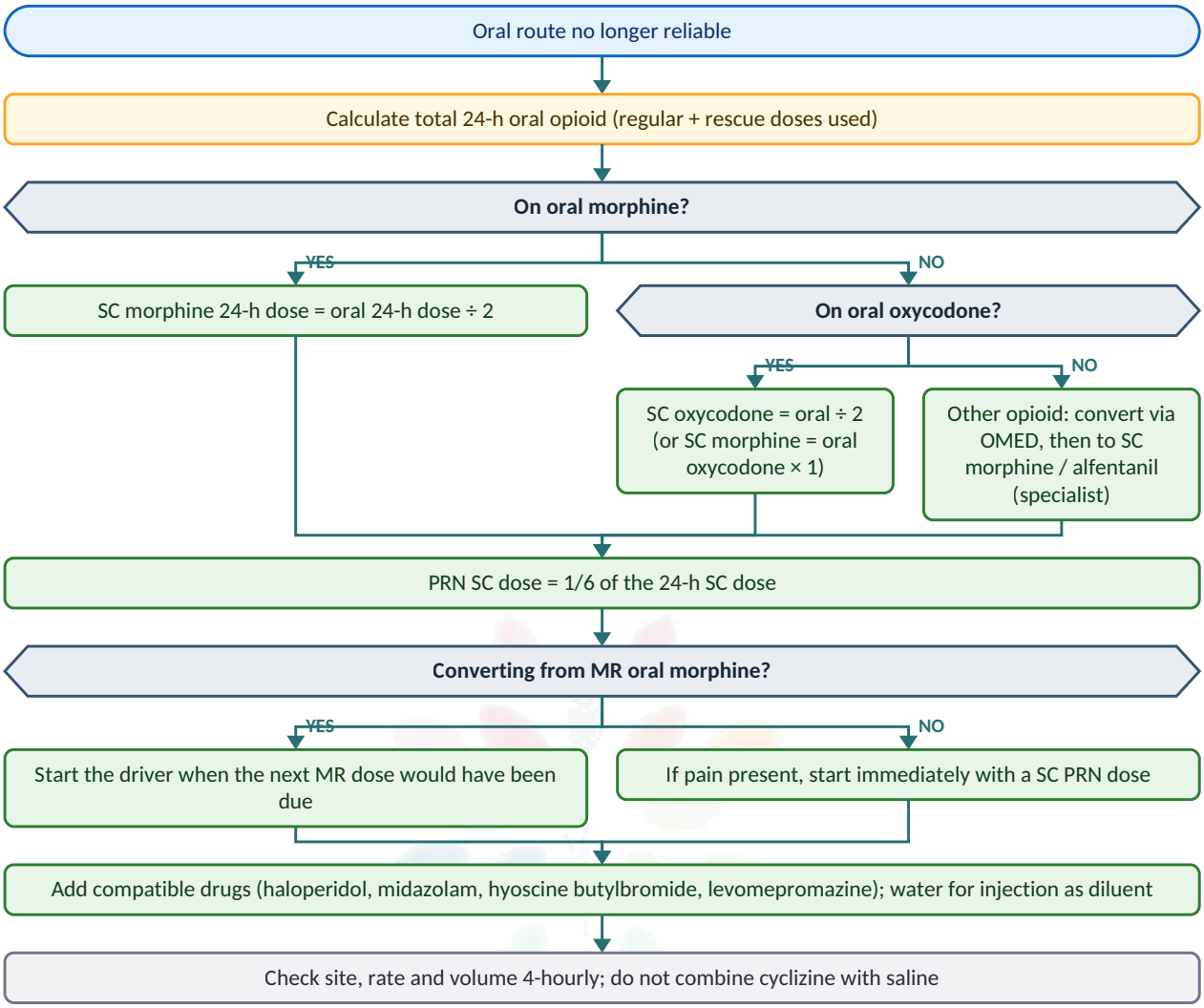
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ALGORITHM ATLAS · Algo 28

Algorithm: Converting to a Syringe Driver (CSCI)

When the oral route fails (dysphagia, vomiting, weakness, dying).

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Fentanyl patches should be continued, not converted, at end of life — add CSCI for extra needs.
- Driver takes ~4 h to reach steady state.

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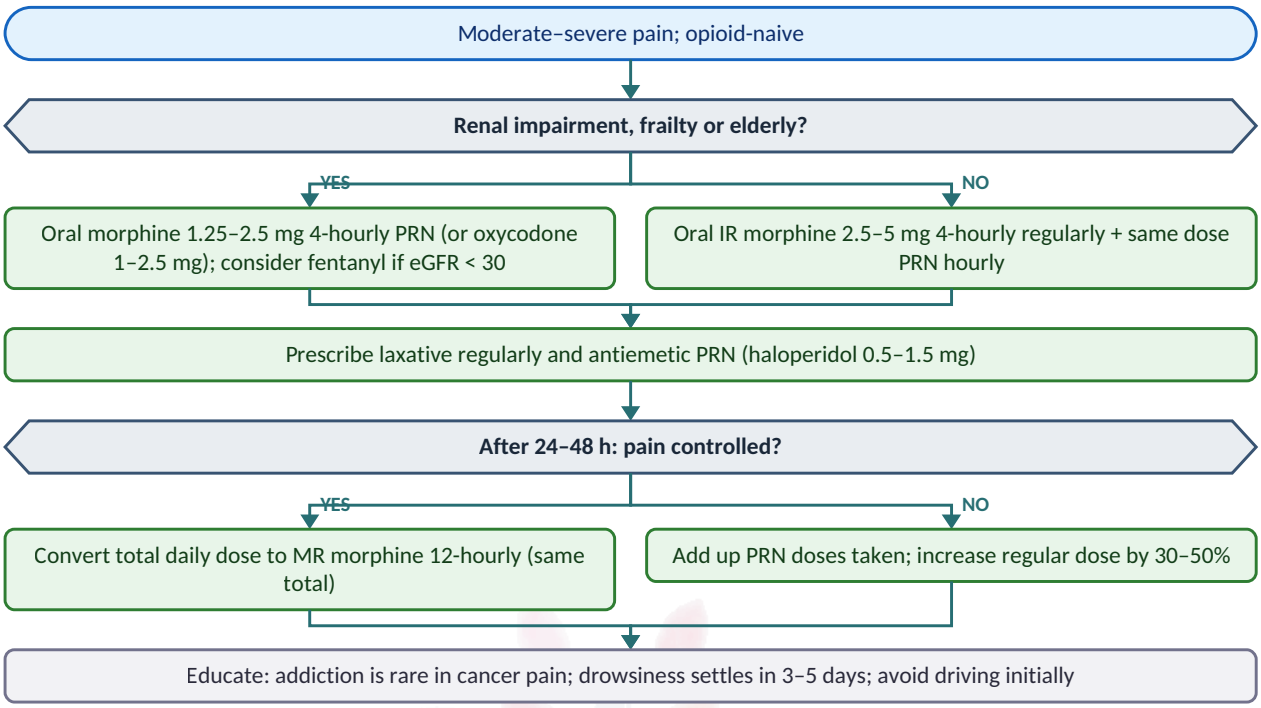
ALGORITHM ATLAS · Algo 29

Algorithm: Starting Opioids in an Opioid-Naive Patient

Read the text ›

Moderate–severe cancer pain not controlled by non-opioids.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Weak opioids (step 2) can be skipped — low-dose morphine is as effective (Bandieri 2016).
- Oral morphine bioavailability ~30%.

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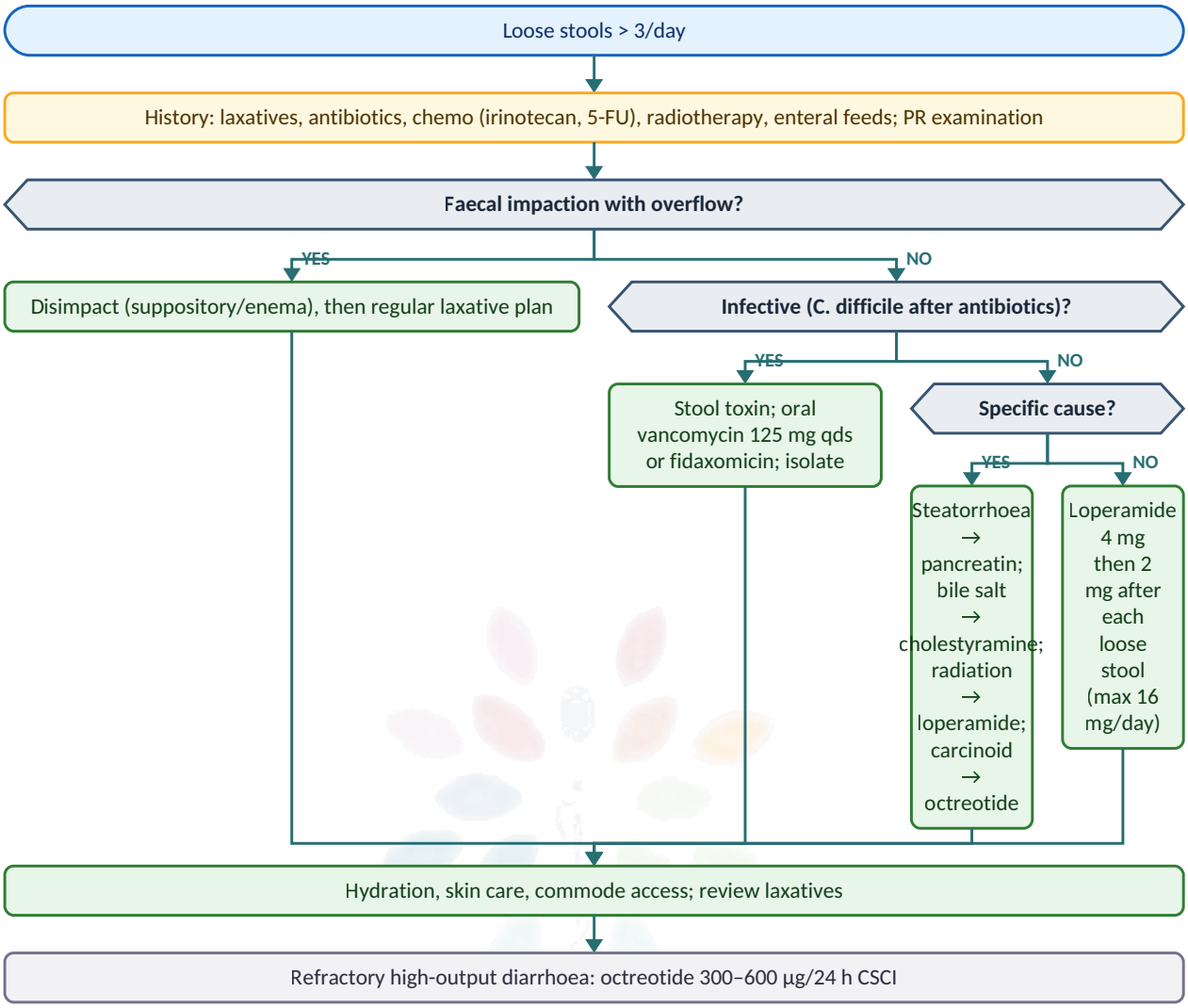
ALGORITHM ATLAS · Algo 30

Algorithm: Diarrhoea in Palliative Care

Read the text ›

Exclude overflow from faecal impaction before treating.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Never start loperamide without excluding impaction and C. difficile.
- Irinotecan early diarrhoea is cholinergic — atropine responds.

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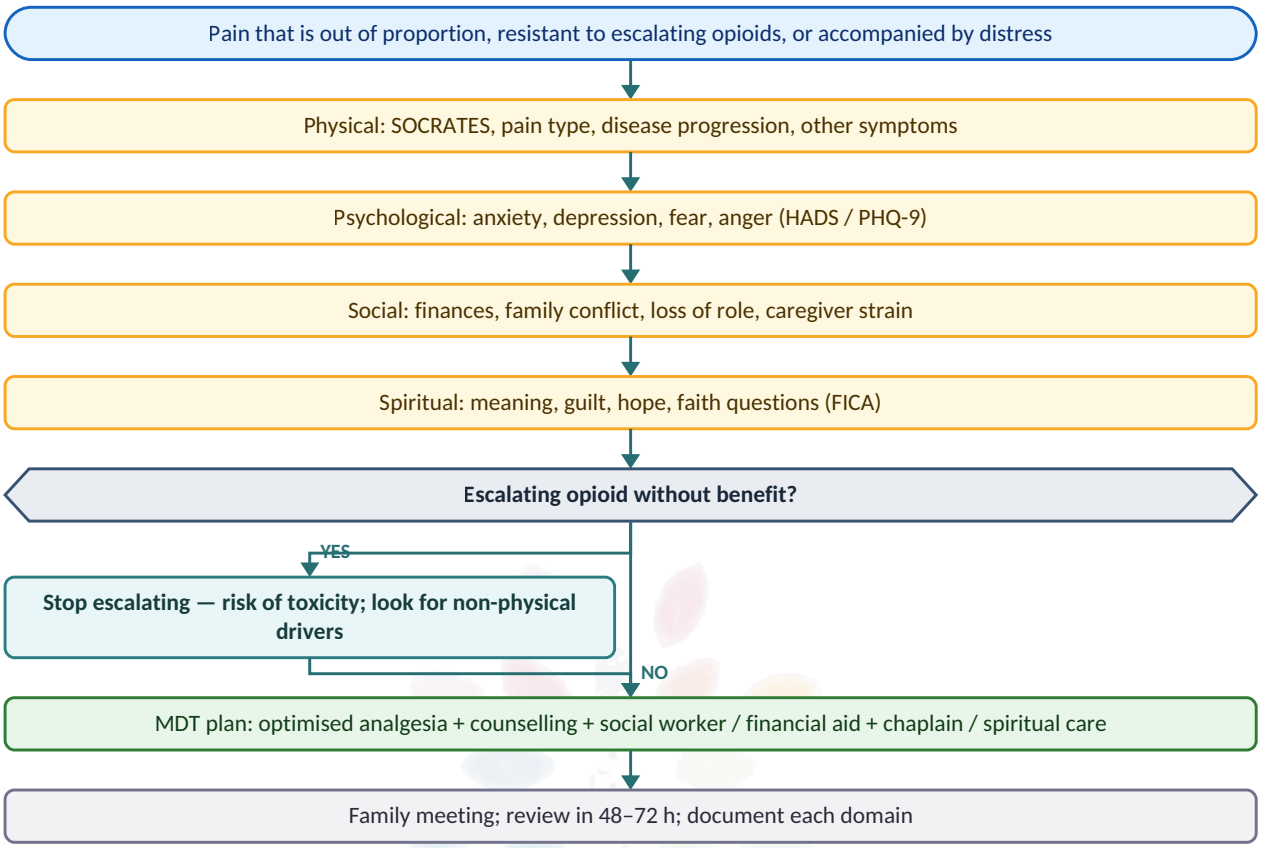
ALGORITHM ATLAS · Algo 31

Algorithm: Assessment and Management of Total Pain

Read the text ›

Cicely Saunders' concept: physical, psychological, social and spiritual suffering combine into one experience of pain.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Chemical coping (using opioids for emotional distress) signals unaddressed total pain.
- Ask: 'What worries you most?' — often reveals the hidden domain.

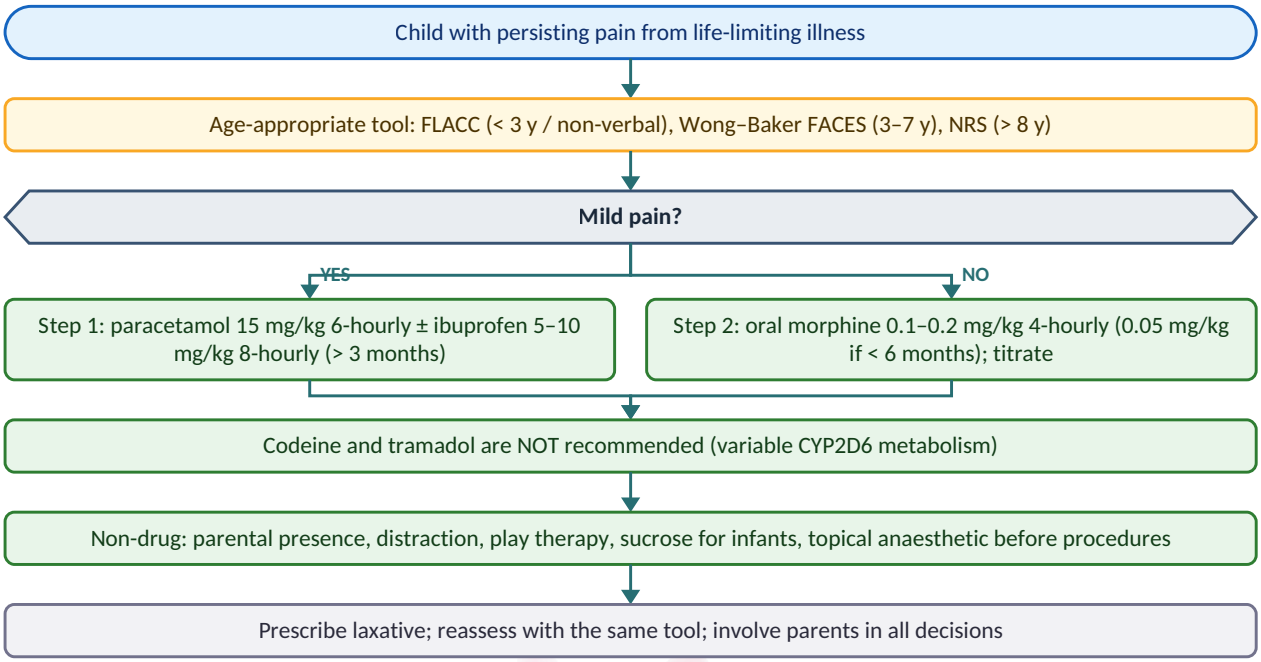
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ALGORITHM ATLAS · Algo 32

Algorithm: Pain Management in Children

WHO (2012) recommends a two-step approach for persisting pain in children.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Children are not small adults — dose by weight and age.
- ACT categories 1–4 classify life-limiting conditions for paediatric palliative care.

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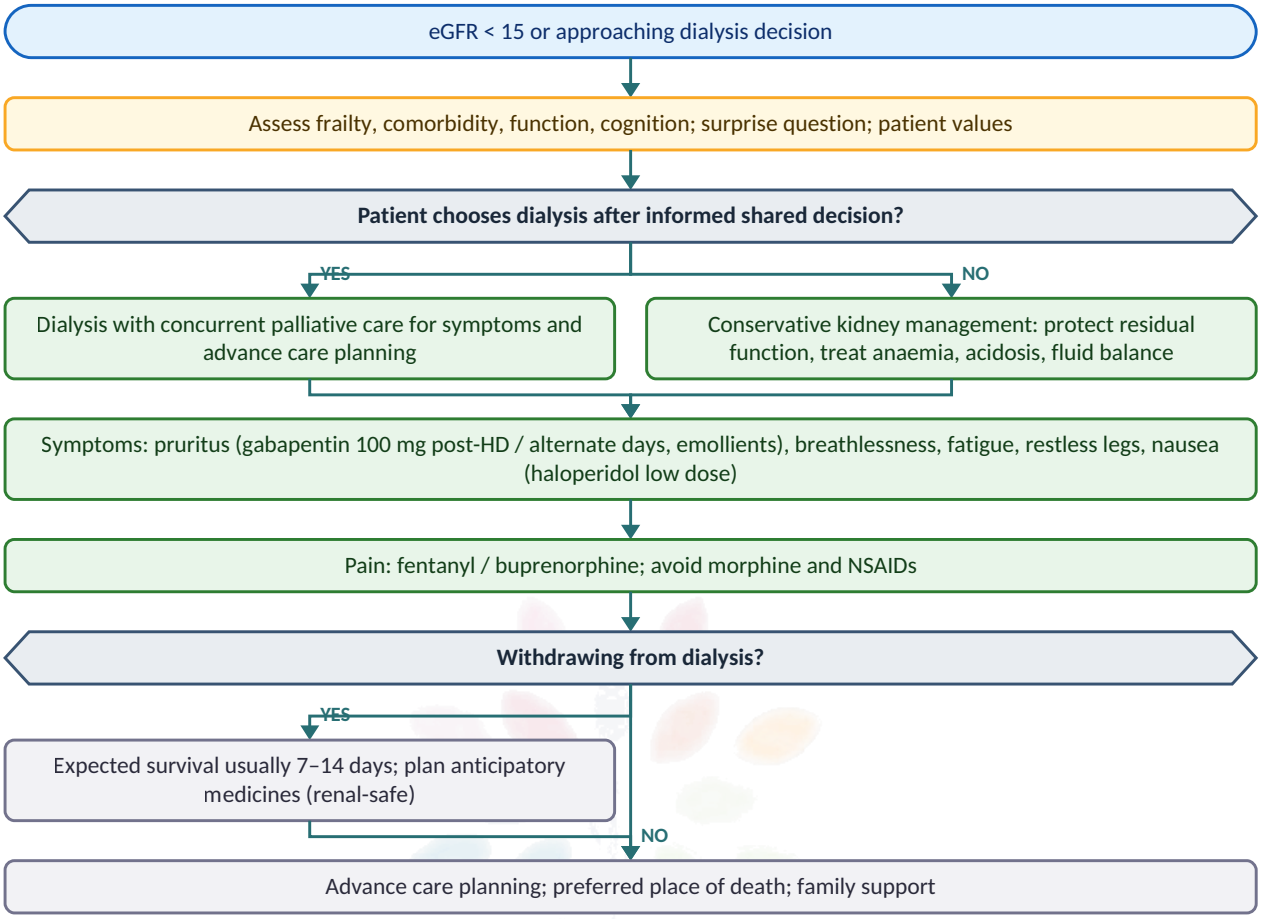
ALGORITHM ATLAS · Algo 33

Algorithm: Conservative Kidney Management (End-Stage Kidney Disease)

Read the text ›

For older, frail or multimorbid patients, dialysis may not prolong life or improve quality.

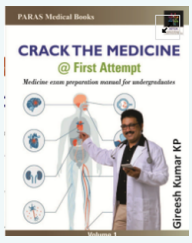
Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Over-80s with high comorbidity may survive as long on conservative management as on dialysis.
- Midazolam and alfentanil are preferred in the dying renal patient.

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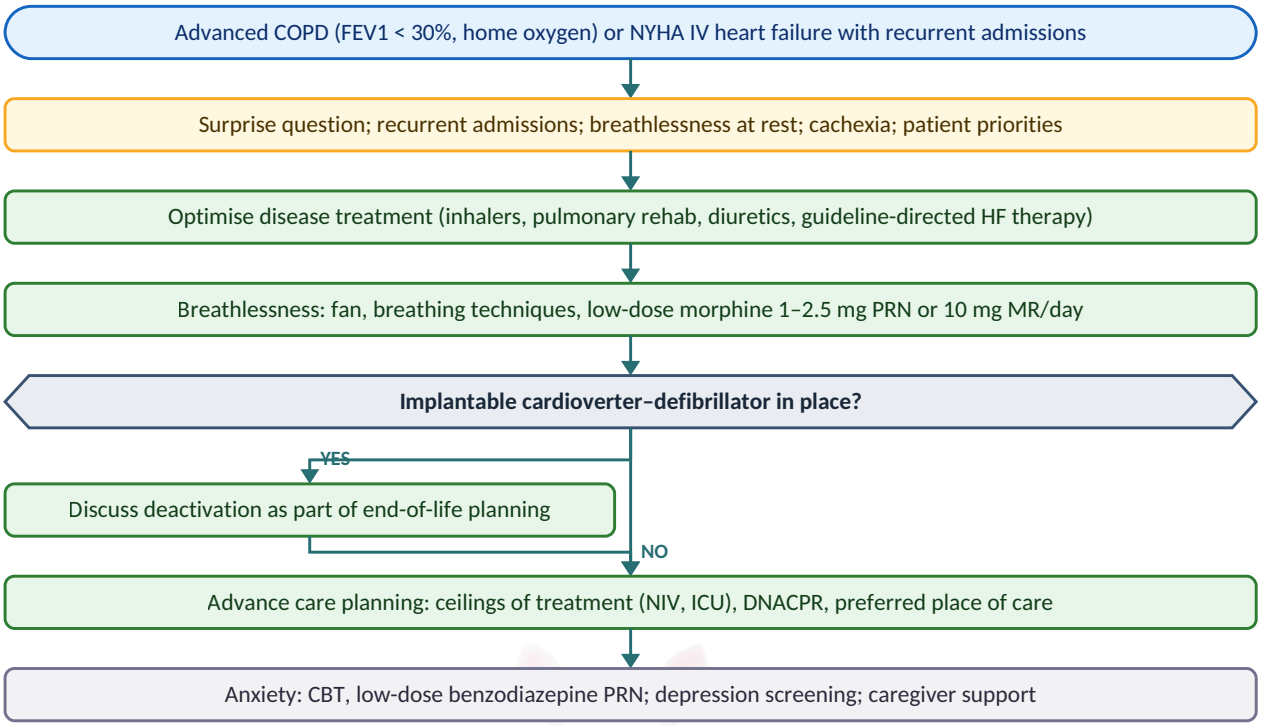
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ALGORITHM ATLAS · Algo 34

Algorithm: End-Stage COPD and Heart Failure

Unpredictable trajectory with acute exacerbations — integrate palliative care early.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Opioids for breathlessness in COPD do not cause clinically important hypercapnia at low doses.
- Prognosis is hard to predict — use triggers, not timelines.

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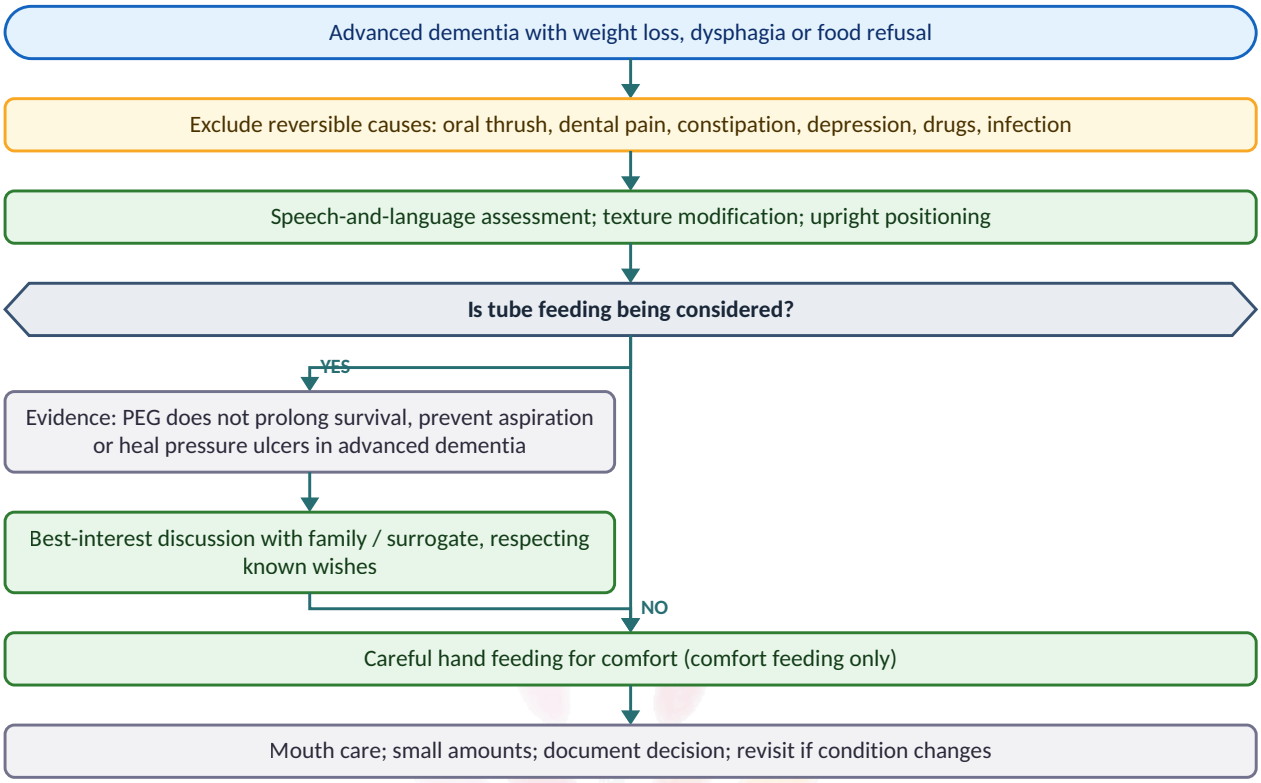
ALGORITHM ATLAS · Algo 35

Algorithm: Feeding Decisions in Advanced Dementia

Read the text ›

Eating difficulties are a marker of end-stage dementia.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Hand feeding offers human contact that tube feeding removes.
- Aspiration pneumonia is common with PEG too (reflux of feeds).

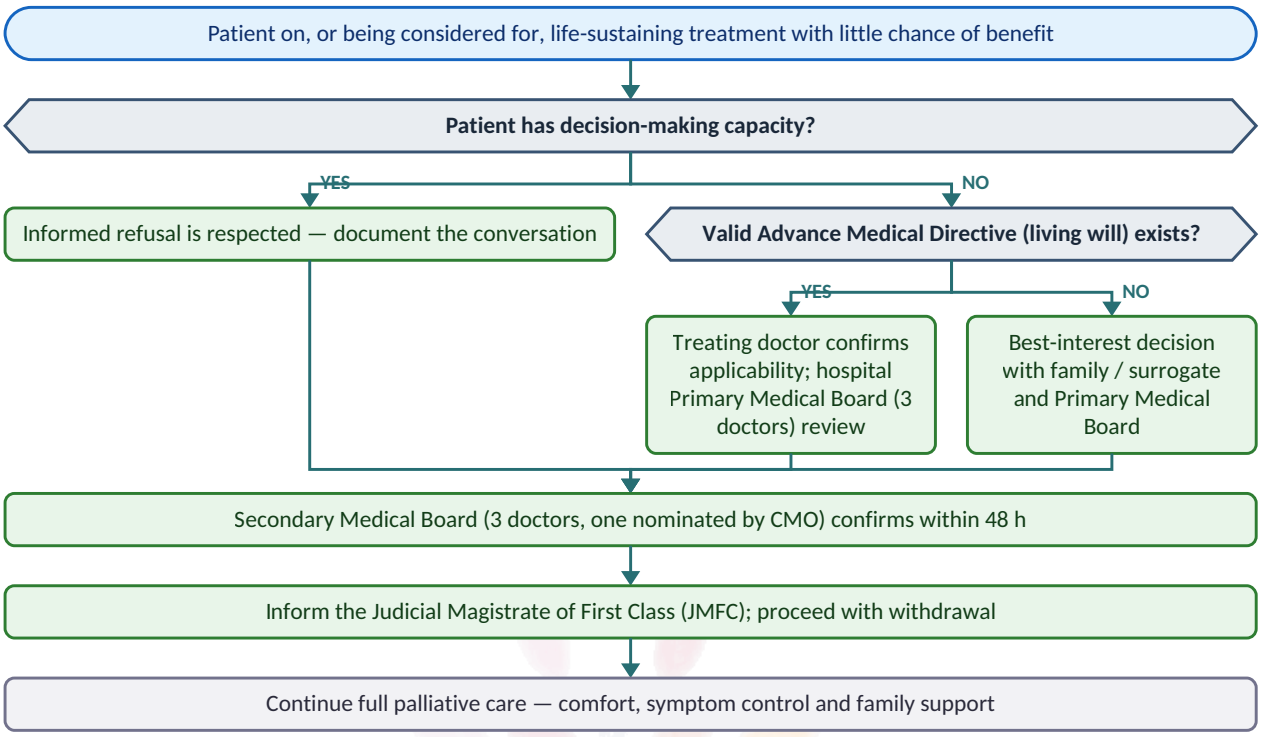
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ALGORITHM ATLAS · Algo 36

Algorithm: Withholding or Withdrawing Life-Sustaining Treatment in India

Common Cause vs Union of India (2018), with procedure simplified by the Supreme Court in 2023.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Withdrawal of futile treatment is not euthanasia — intent is to stop burdensome treatment, not to cause death.
- The 2023 modification removed the need for magistrate countersignature of living wills (notary/gazetted officer attestation suffices).

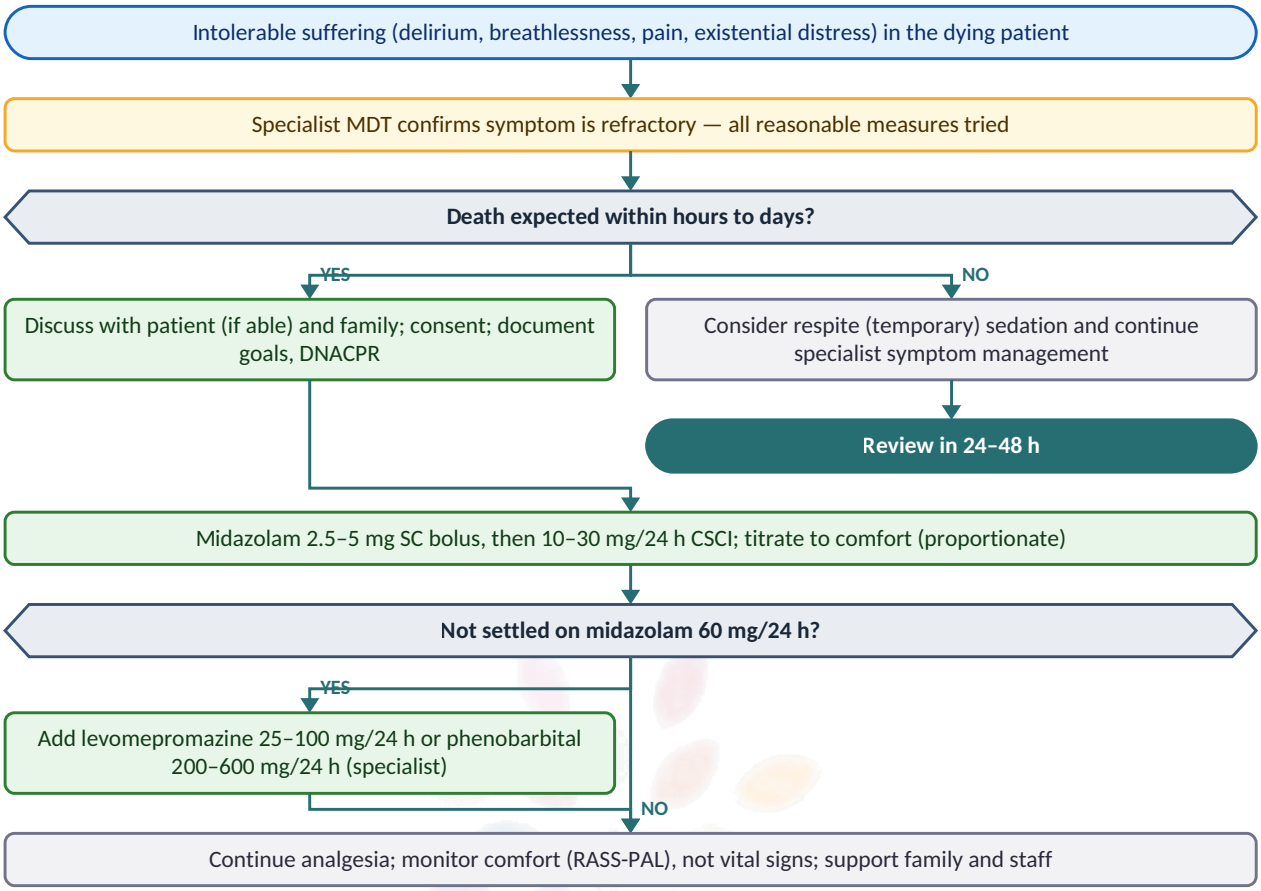
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ALGORITHM ATLAS · Algo 37

Algorithm: Palliative Sedation

Proportionate lowering of consciousness to relieve refractory suffering in the last days.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Proportionate sedation does not hasten death when appropriately titrated (Maltoni 2012).
- Doctrine of double effect: intent is relief of suffering.

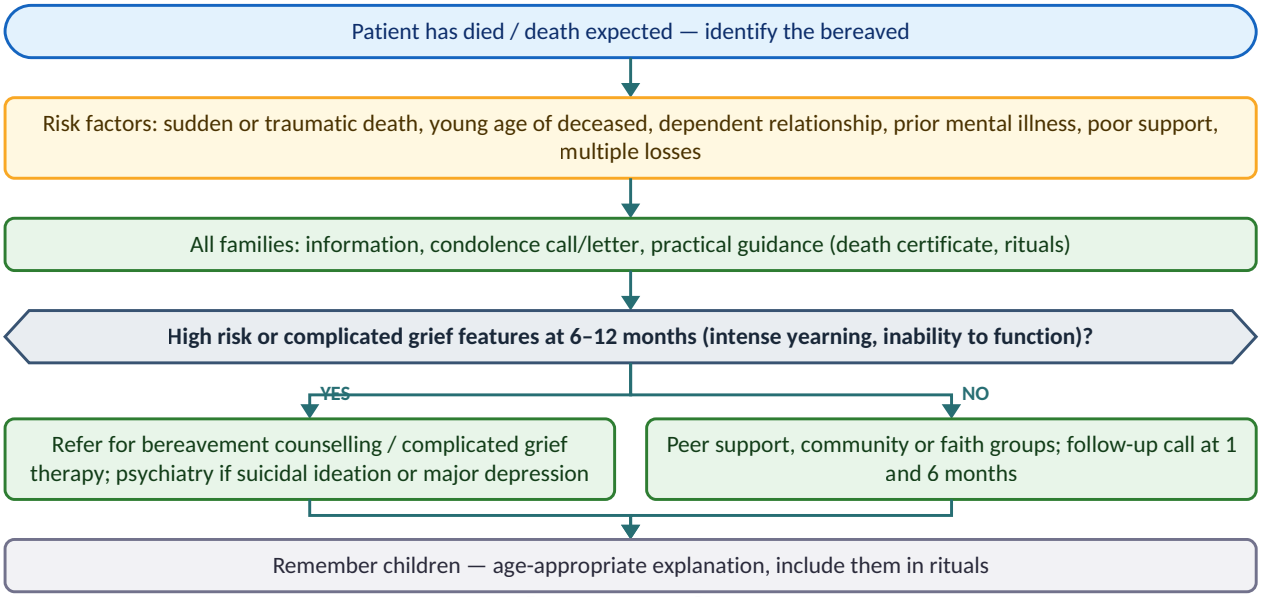
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ALGORITHM ATLAS · Algo 38

Algorithm: Bereavement Risk Assessment and Support

Most grief is normal and needs information and support, not therapy.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Prolonged grief disorder (ICD-11): persistent grief > 6 months with functional impairment.
- Kübler-Ross stages are not linear — dual process model is more useful.

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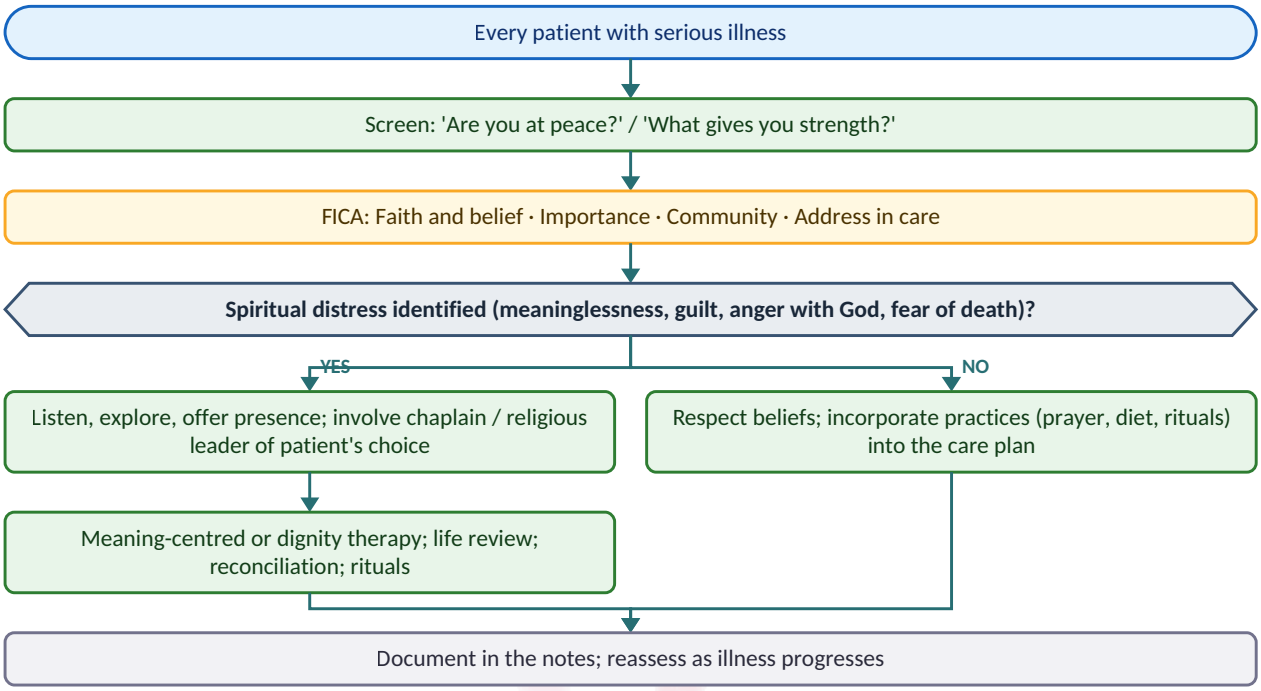
ALGORITHM ATLAS · Algo 39

Algorithm: Spiritual Assessment (FICA) and Care

Read the text ›

Spiritual distress amplifies pain and anxiety; screening is everyone's job.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Spirituality is broader than religion — it is about meaning and connection.
- HOPE and SPIRIT are alternative assessment tools.

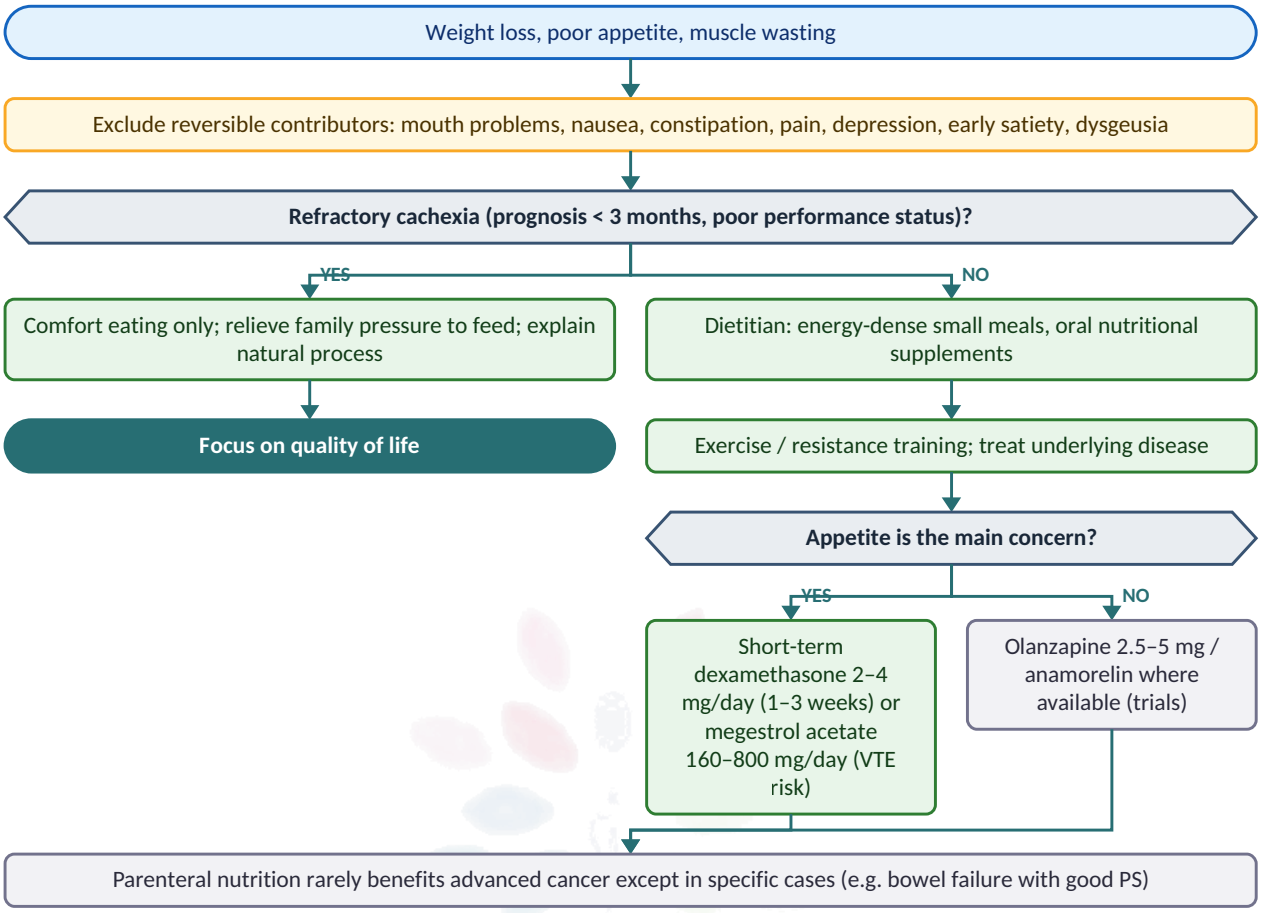
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ALGORITHM ATLAS · Algo 40

Algorithm: Cancer Anorexia–Cachexia Syndrome

Fearon 2011: weight loss > 5% in 6 months (or > 2% with BMI < 20 / sarcopenia); pre-cachexia → cachexia → refractory cachexia.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Cachexia is driven by inflammation (IL-6, TNF-α) — not simply starvation.
- Family distress over 'not eating' is a major issue — address it explicitly.

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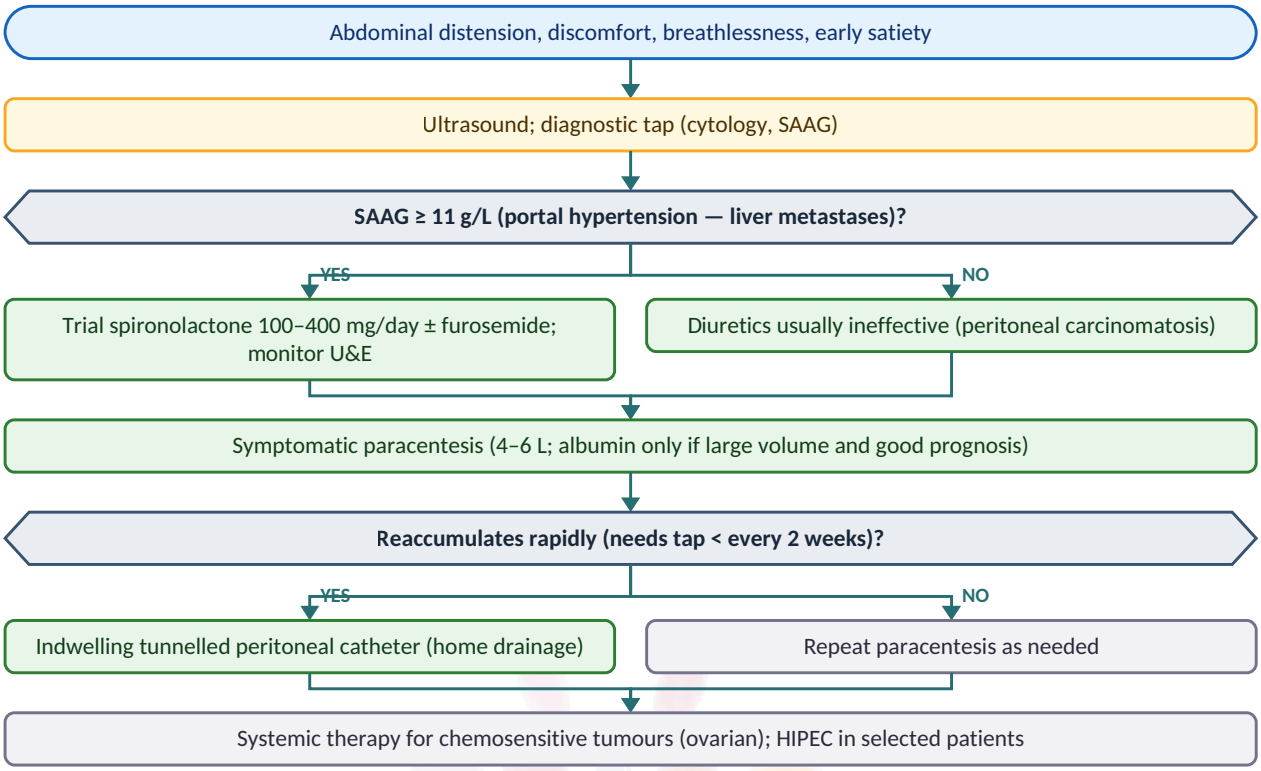
ALGORITHM ATLAS · Algo 41

Algorithm: Malignant Ascites

Read the text ›

Common with ovarian, GI, breast cancers and lymphoma.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Malignant ascites median survival is often < 6 months — discuss goals.
- Catumaxomab (EpCAM) was used in Europe for EpCAM+ ascites.

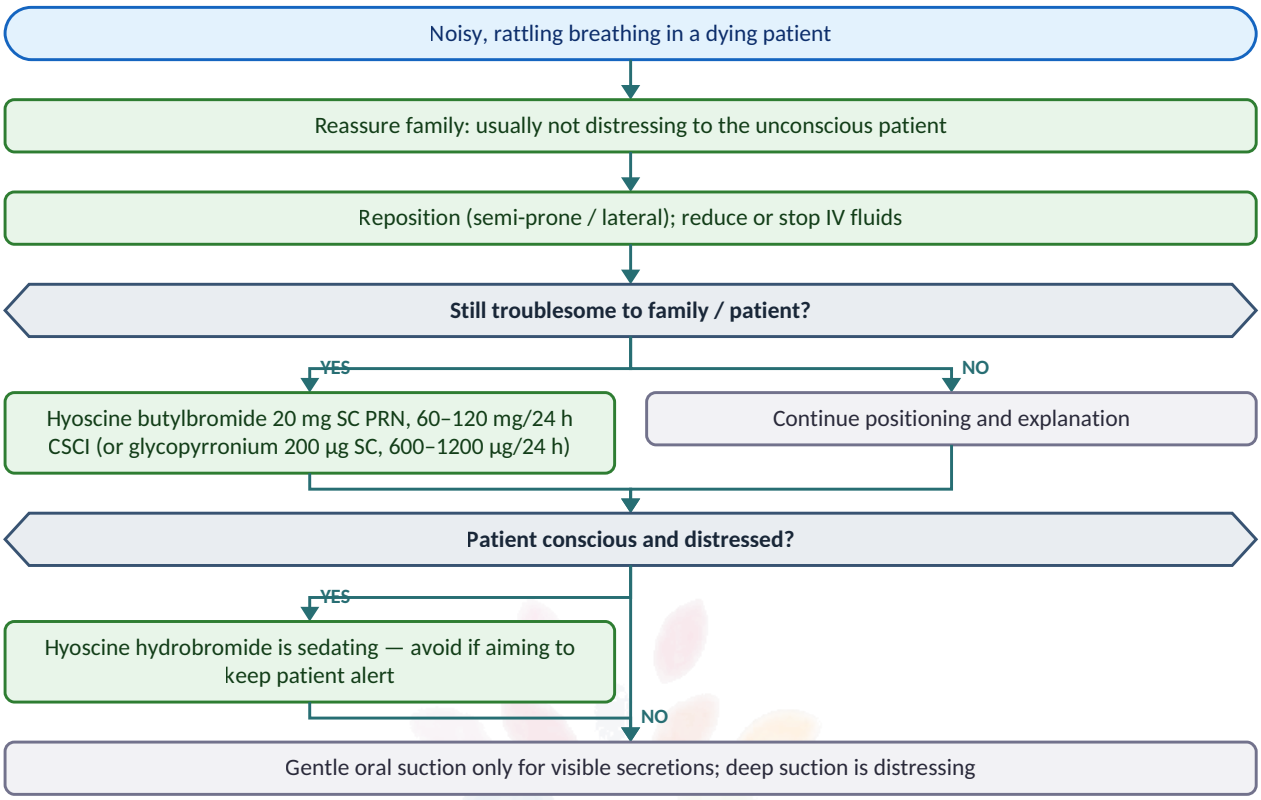
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ALGORITHM ATLAS · Algo 42

Algorithm: Terminal Respiratory Secretions (Death Rattle)

Noisy breathing from pooled secretions in the last hours–days; occurs in ~40% of dying patients.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Anticholinergics prevent new secretions but do not clear existing ones — start early.
- Evidence for antimuscarinics is weak (Cochrane) — explanation is the core intervention.

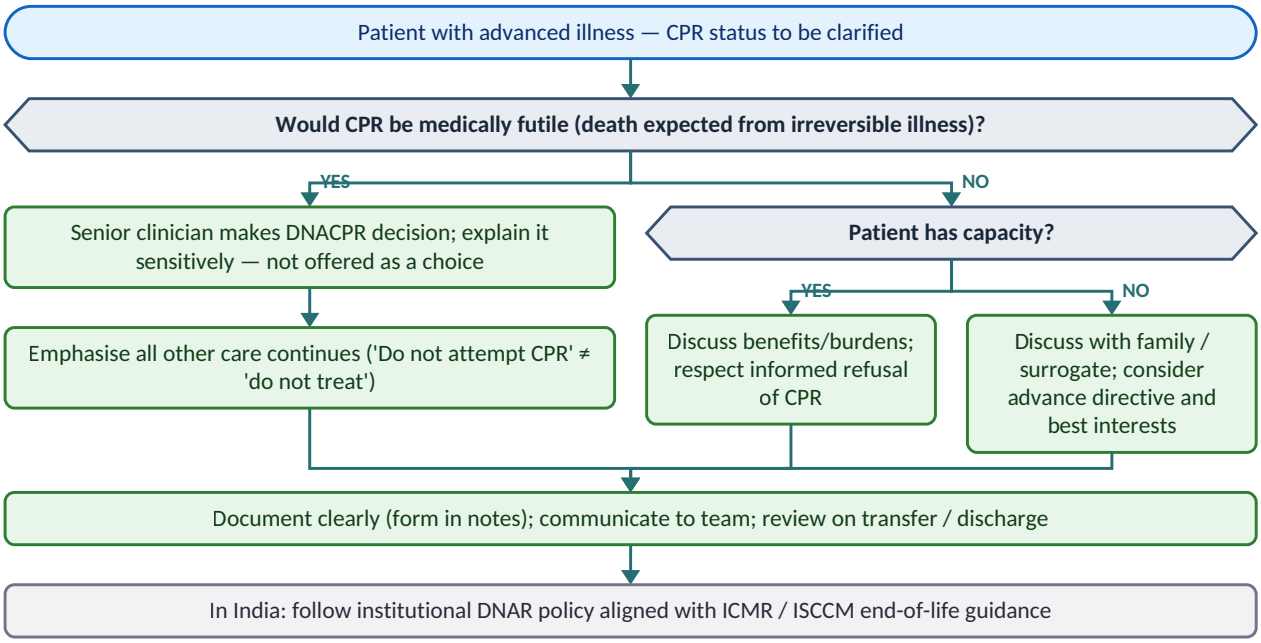
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ALGORITHM ATLAS · Algo 43

Algorithm: Do-Not-Attempt CPR (DNACPR) Decision

CPR has a survival-to-discharge rate of < 5% in advanced metastatic cancer.

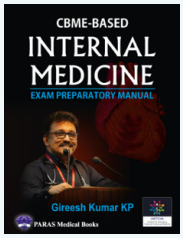
Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- 'Allow natural death' language is better understood by families.
- DNACPR does not mean withholding antibiotics, fluids or symptom control.

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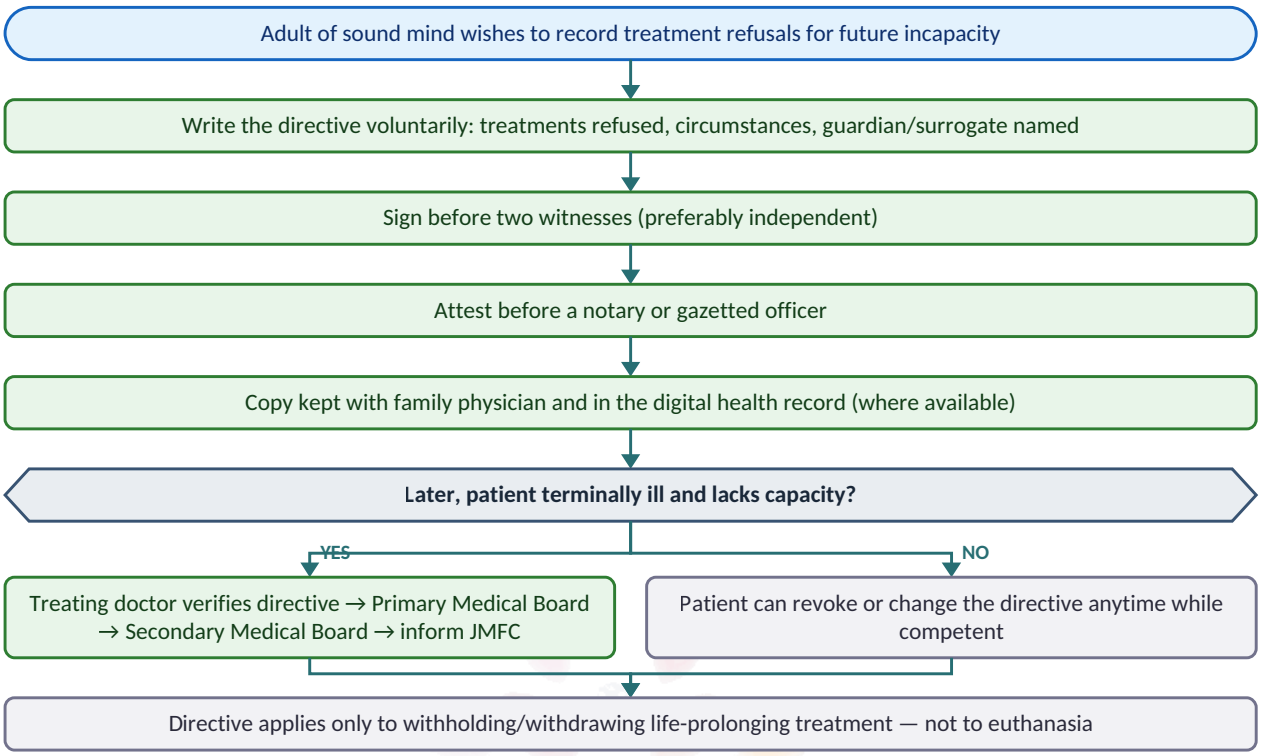
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ALGORITHM ATLAS · Algo 44

Algorithm: Making an Advance Medical Directive in India

Supreme Court procedure as simplified in January 2023.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Advance care planning conversations matter more than the form itself.
- Name a trusted surrogate who knows the patient's values.

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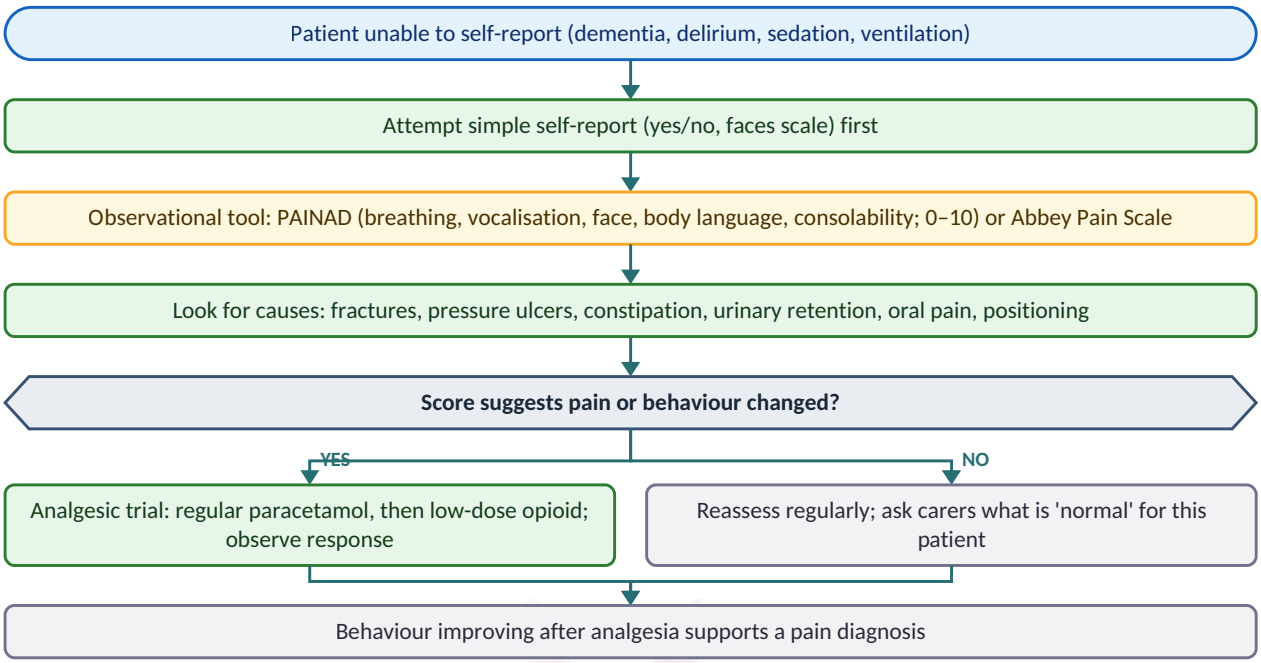
ALGORITHM ATLAS · Algo 45

Algorithm: Pain Assessment in Non-Verbal and Cognitively Impaired Patients

Read the text ›

Self-report is the gold standard — use observational scales when it is not possible.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Agitation in dementia is often unrecognised pain.
- Critical-care Pain Observation Tool (CPOT) for ventilated patients.

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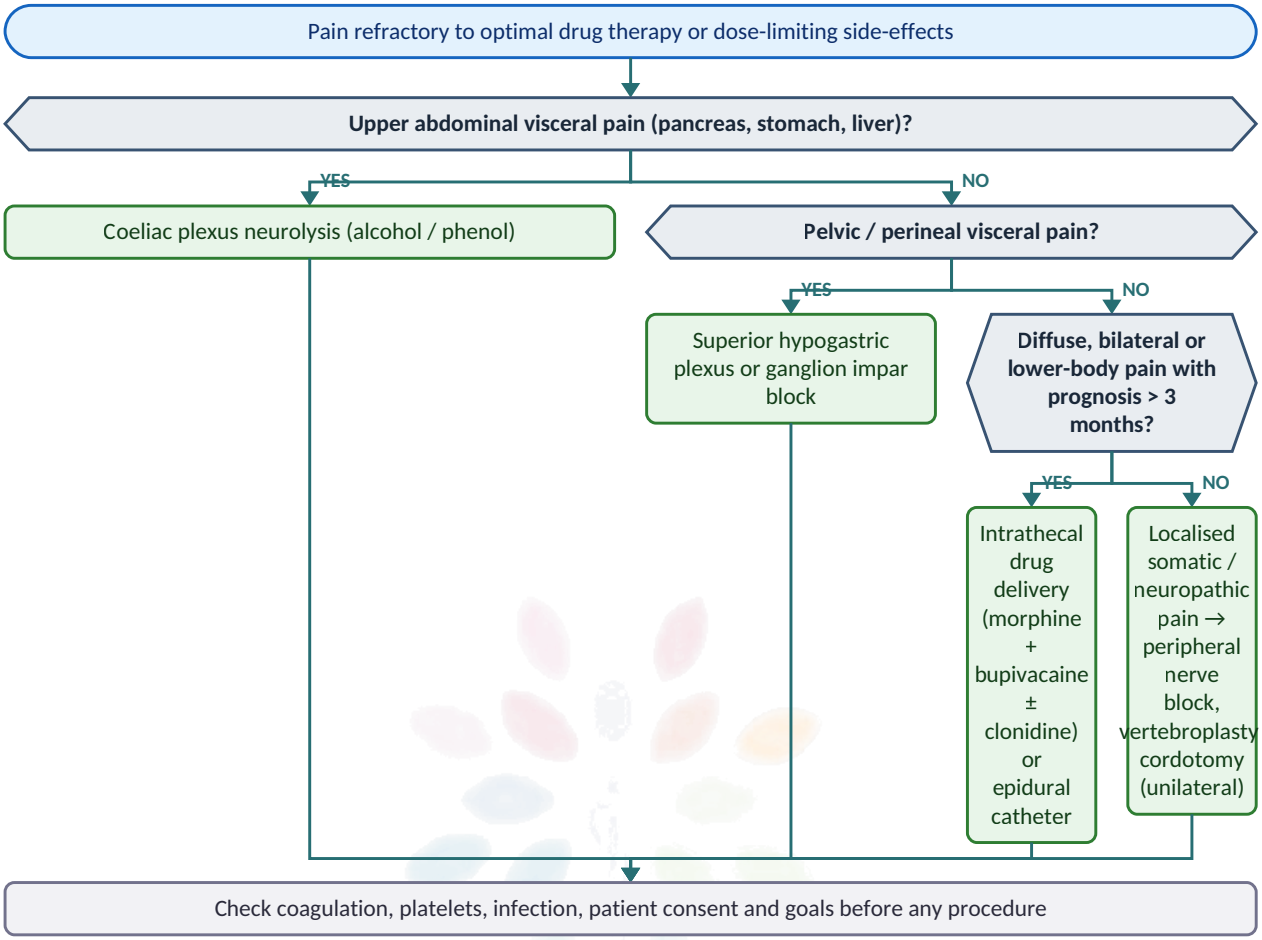
ALGORITHM ATLAS · Algo 46

Algorithm: Choosing an Interventional Pain Technique

Read the text ›

About 10–15% of cancer pain needs interventions beyond drugs.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Early coeliac block reduces opioid use and constipation in pancreatic cancer.
- Percutaneous cervical cordotomy suits unilateral mesothelioma pain.

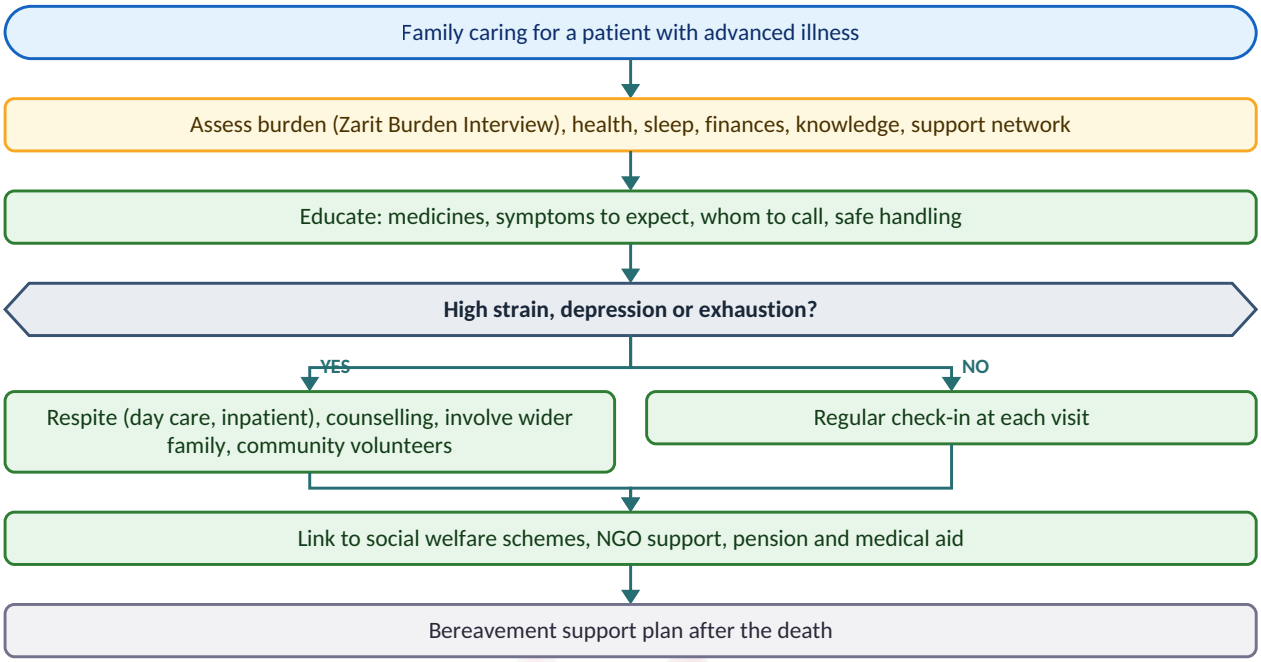
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ALGORITHM ATLAS · Algo 47

Algorithm: Supporting the Family Caregiver

Caregivers in India provide most home care, often with financial strain.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Caregiver burden predicts complicated grief.
- Ask the caregiver 'How are you coping?' at every visit.

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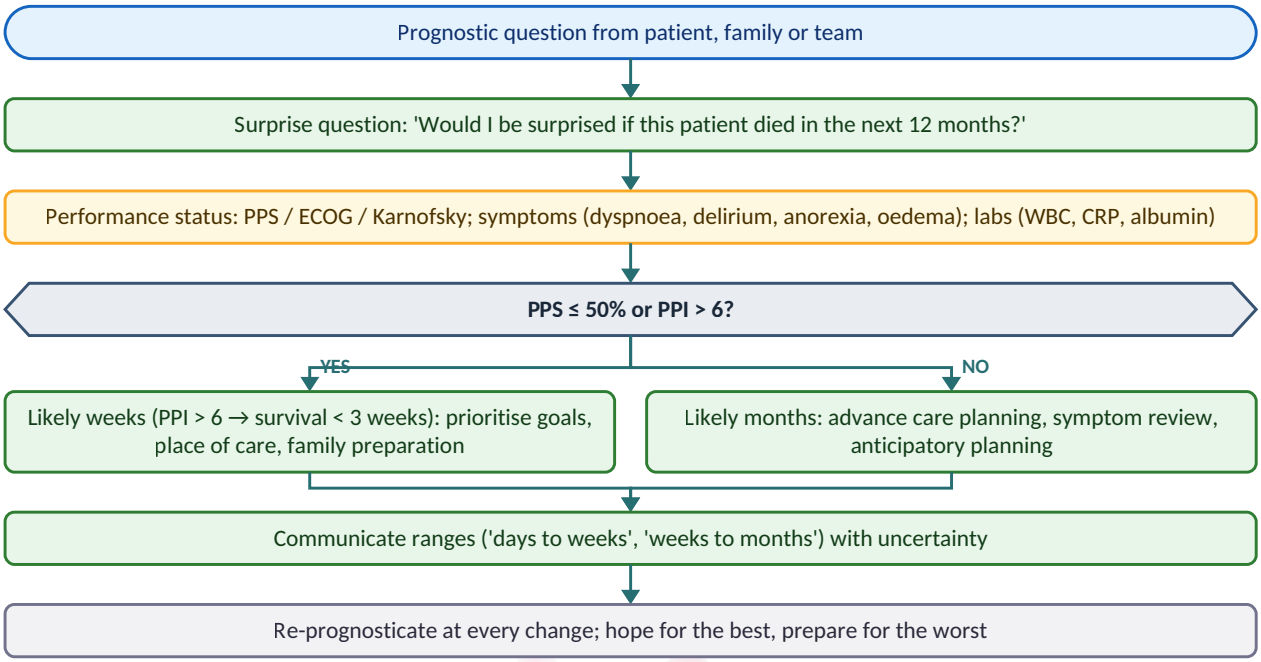
ALGORITHM ATLAS · Algo 48

Algorithm: Prognostication in Advanced Illness

Read the text ›

Clinicians overestimate survival by a factor of 3–5; combine clinical judgment with tools.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- PaP score uses dyspnoea, anorexia, KPS, CPS, WBC and lymphocyte %.
- Rate of change in function is a powerful prognostic clue.

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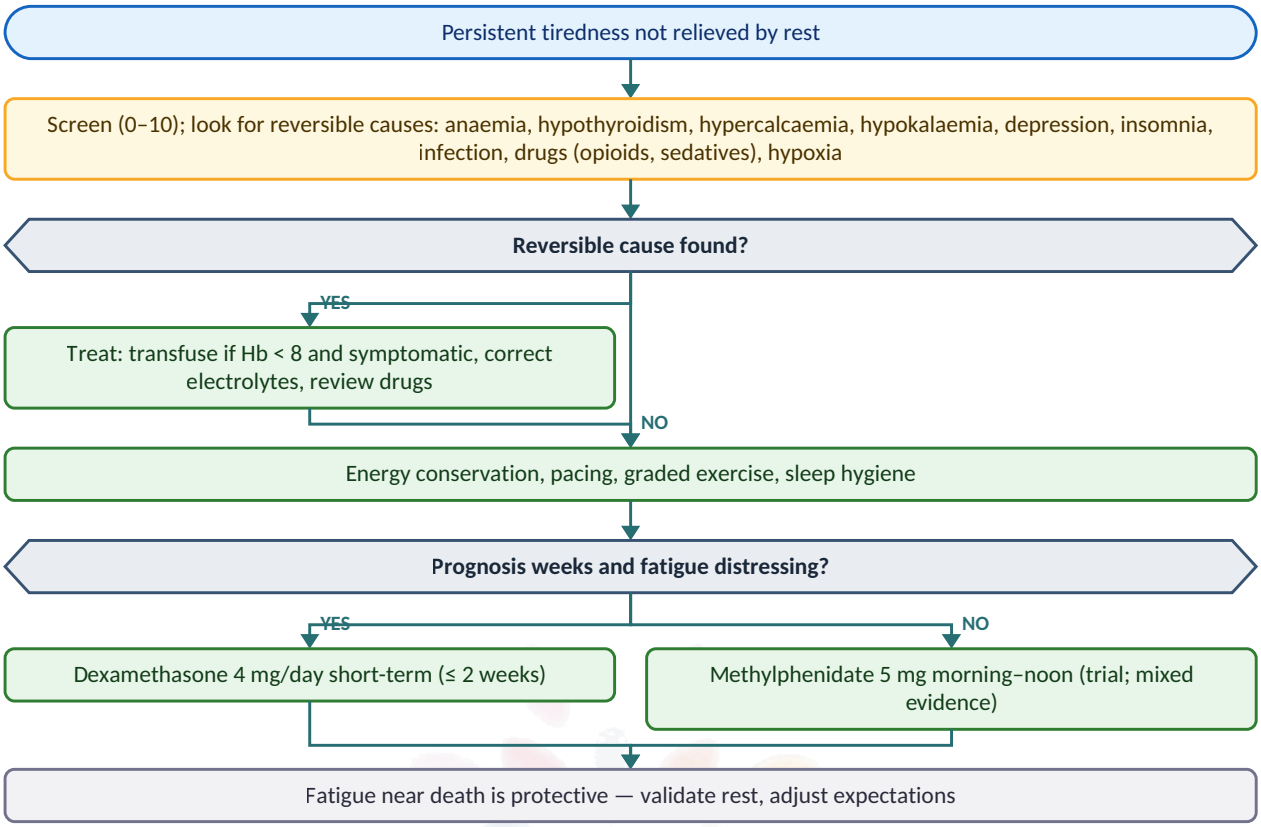
ALGORITHM ATLAS · Algo 49

Algorithm: Fatigue in Palliative Care

Read the text ›

The commonest symptom in advanced cancer.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Exercise has the strongest evidence for cancer-related fatigue in earlier disease.
- Steroid benefit wears off after 2–4 weeks.

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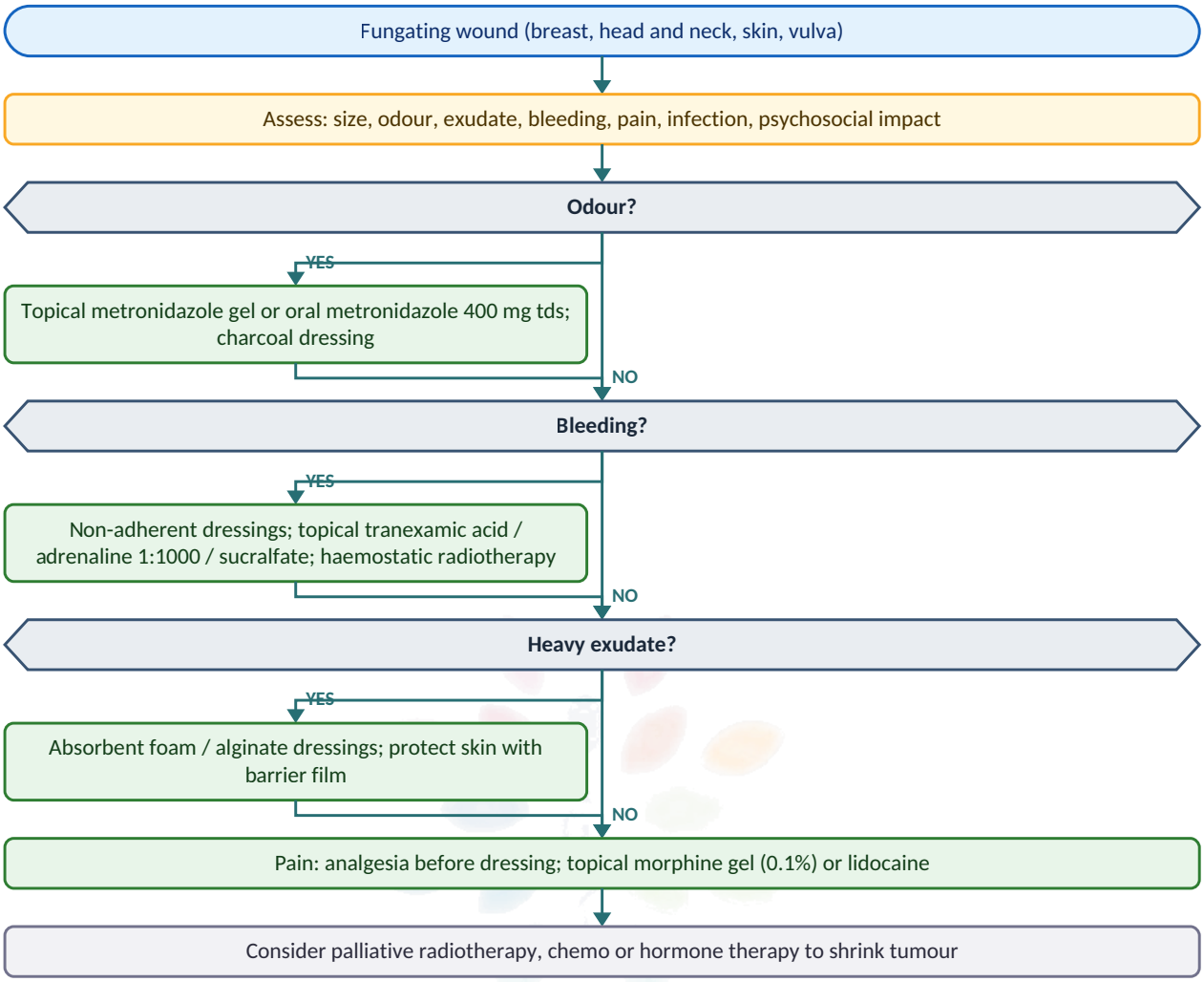
ALGORITHM ATLAS · Algo 50

Algorithm: Malignant Fungating Wound Care

Read the text ›

Tumour infiltration of skin — aim is comfort, not healing.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Odour is the most distressing feature for patients and families.
- Change dressings gently — irrigate rather than scrub.

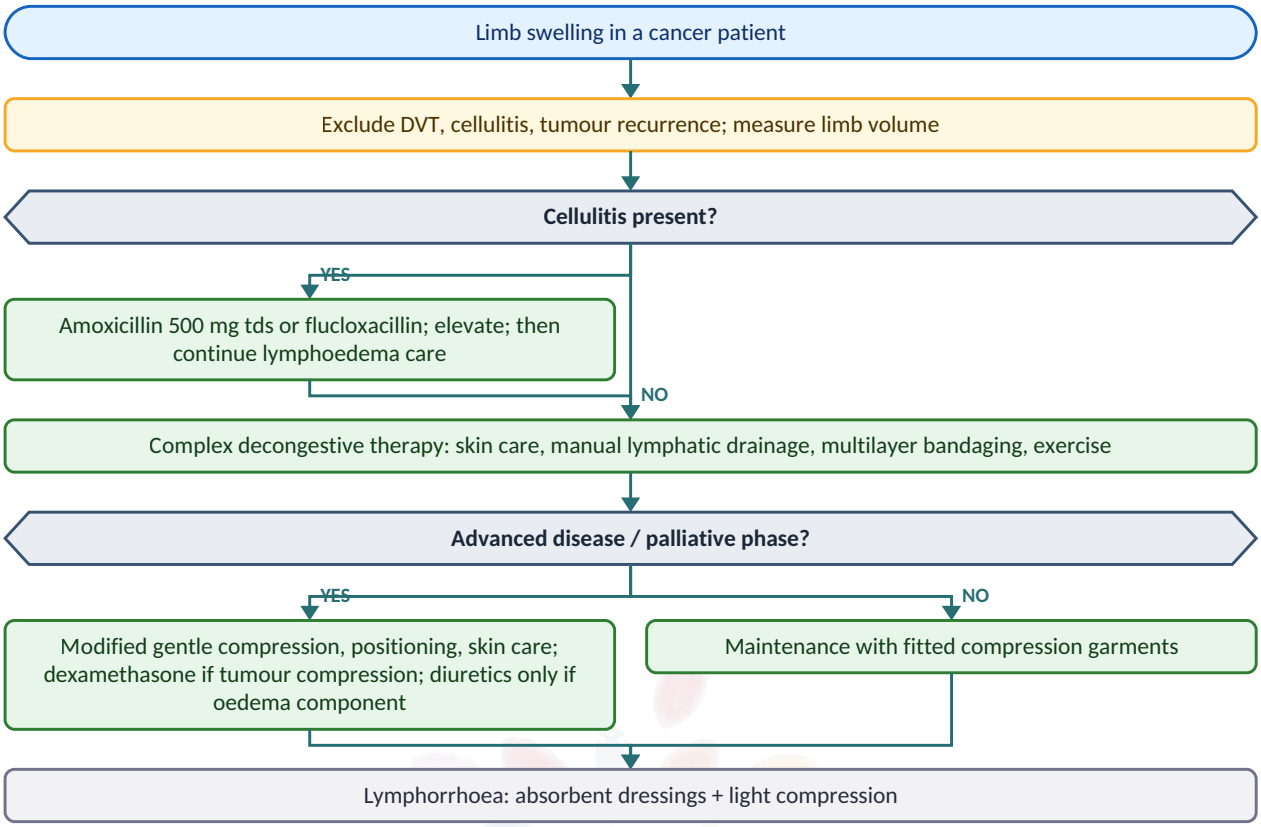
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ALGORITHM ATLAS · Algo 51

Algorithm: Lymphoedema Management

Chronic swelling from lymphatic failure after surgery, radiotherapy or tumour obstruction.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Lymphoedema does not respond to diuretics.
- Skin care prevents cellulitis — the commonest complication.

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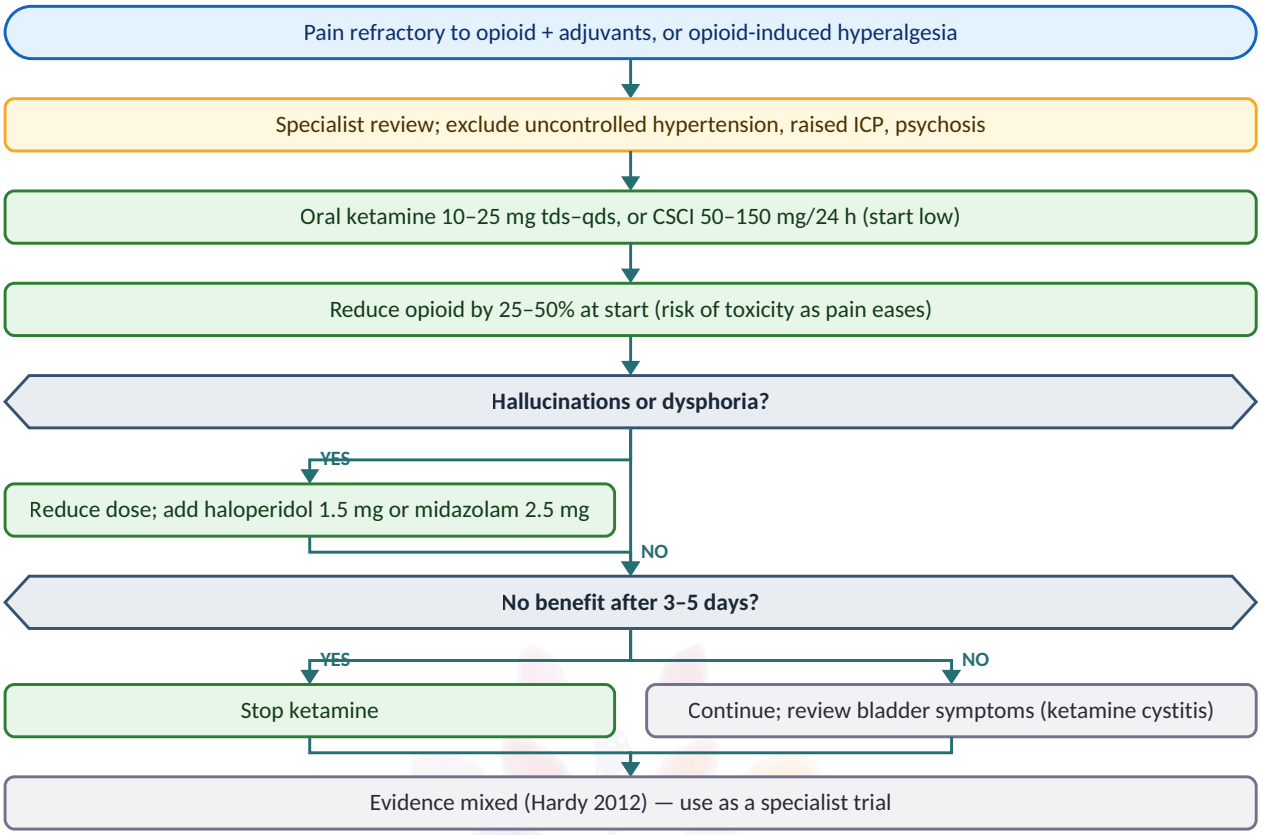
ALGORITHM ATLAS · Algo 52

Algorithm: Ketamine for Refractory Pain

Read the text ›

NMDA antagonist for opioid-resistant neuropathic or hyperalgesic pain (specialist only).

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Sub-anaesthetic doses act on NMDA-mediated central sensitisation.
- Burst ketamine protocols are used in some centres.

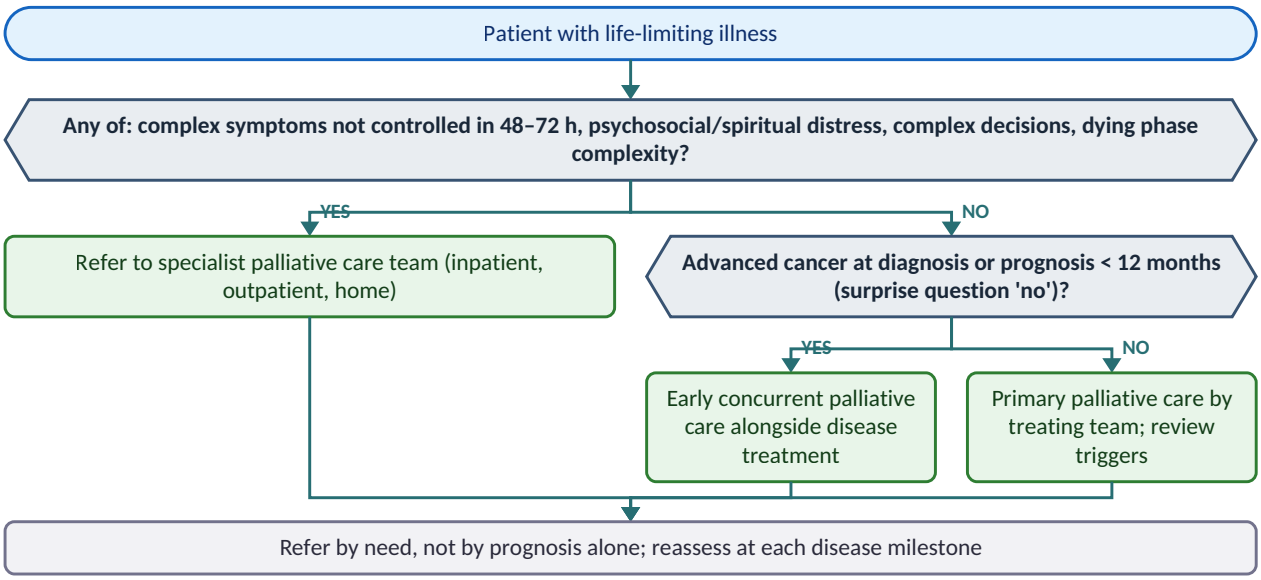
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ALGORITHM ATLAS · Algo 53

Algorithm: When to Refer to Specialist Palliative Care

Early integration improves quality of life (Temel 2010).

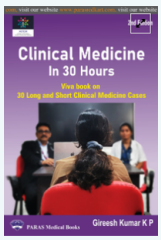
Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- ASCO recommends specialist palliative referral within 8 weeks of advanced cancer diagnosis.
- Palliative care is not only for the dying.

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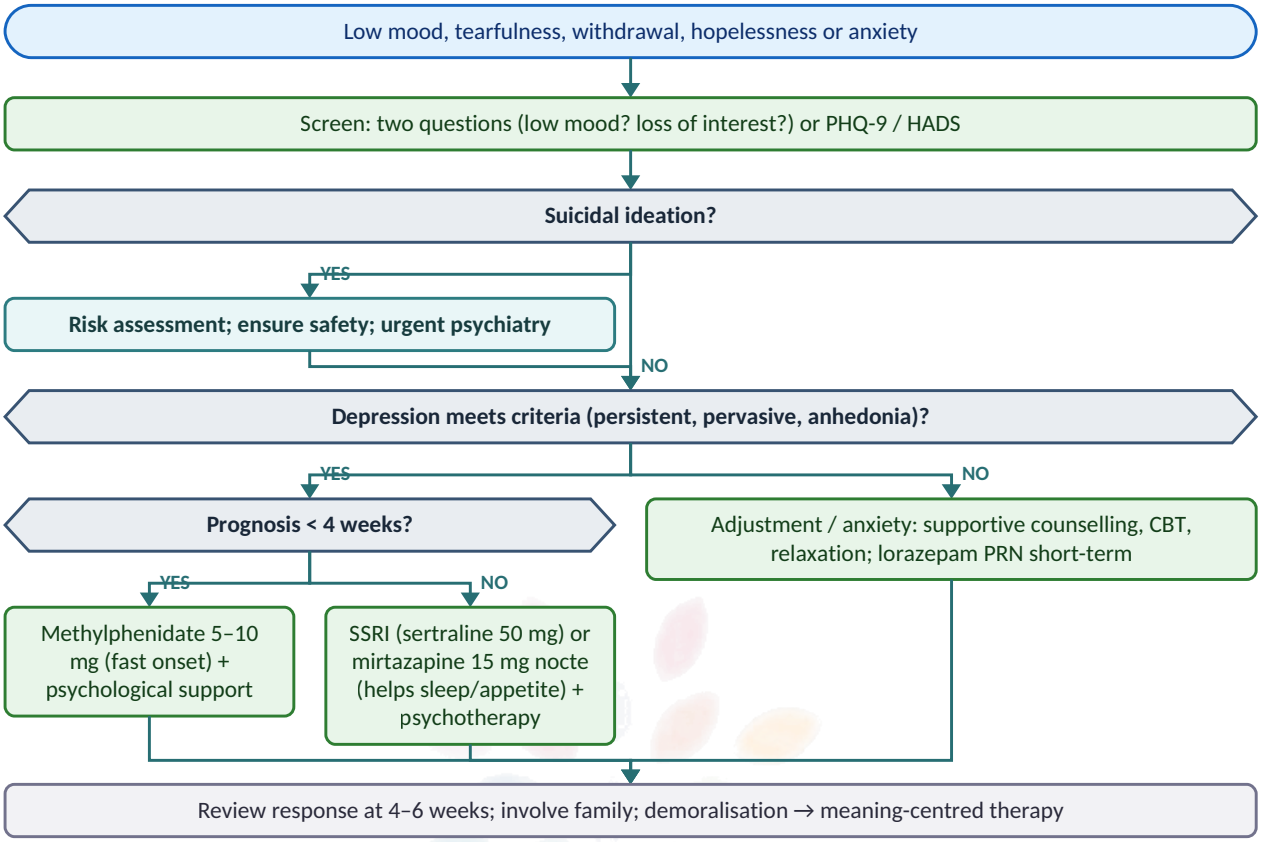
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ALGORITHM ATLAS · Algo 54

Algorithm: Depression and Anxiety in Palliative Care

Depression affects ~15% of patients with advanced cancer and is under-treated.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Somatic symptoms (fatigue, anorexia) are unreliable markers in cancer — focus on cognitive features.
- Mirtazapine is versatile in palliative care.

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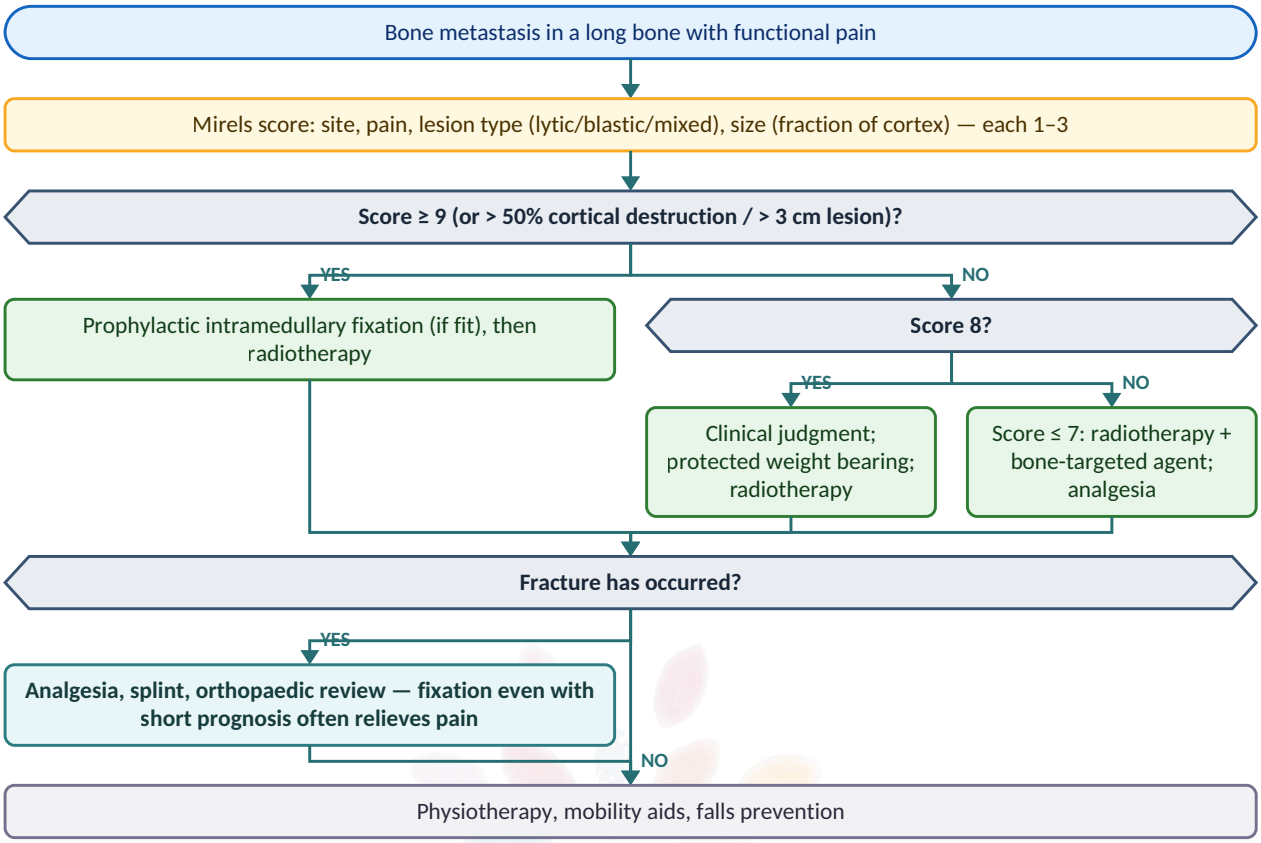
ALGORITHM ATLAS · Algo 55

Algorithm: Impending Pathological Fracture

Read the text ›

Mirels score predicts long-bone fracture risk from metastases.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Hip fracture fixation is usually palliative surgery — prevents agonising pain.
- Spine lesions → assess with SINS (spinal instability score).

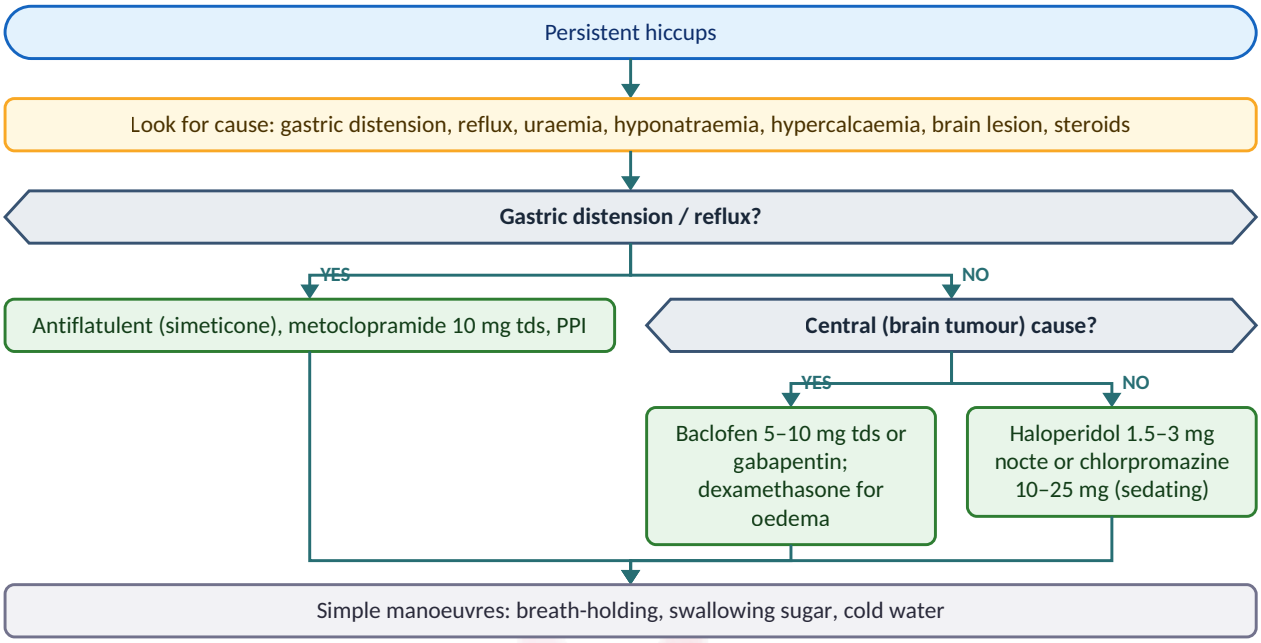
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ALGORITHM ATLAS · Algo 56

Algorithm: Hiccups

Persistent hiccups (> 48 h) are exhausting; causes include gastric distension, phrenic irritation, uraemia and CNS lesions.

Start · Assess · Decision · Action · Urgent · Info · Outcome



★ KEY POINTS

- Dexamethasone can itself cause hiccups.
- Baclofen is effective for central and peripheral hiccups.

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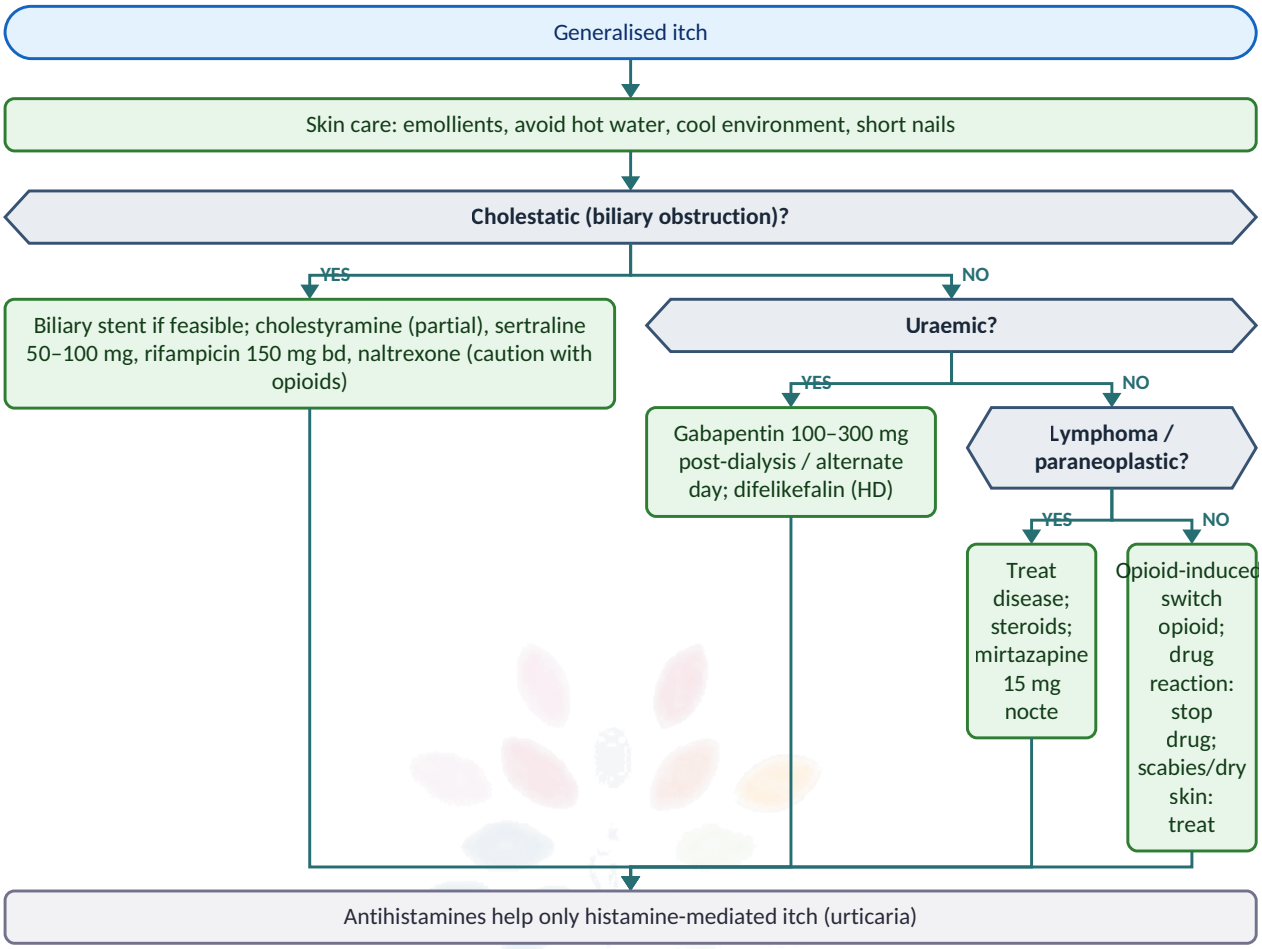
ALGORITHM ATLAS · Algo 57

Algorithm: Pruritus in Palliative Care

Read the text ›

Itch has different mechanisms — treat by cause.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Opioid antagonists can precipitate pain and withdrawal in patients on opioids.
- Sertraline and mirtazapine are useful in mixed causes.

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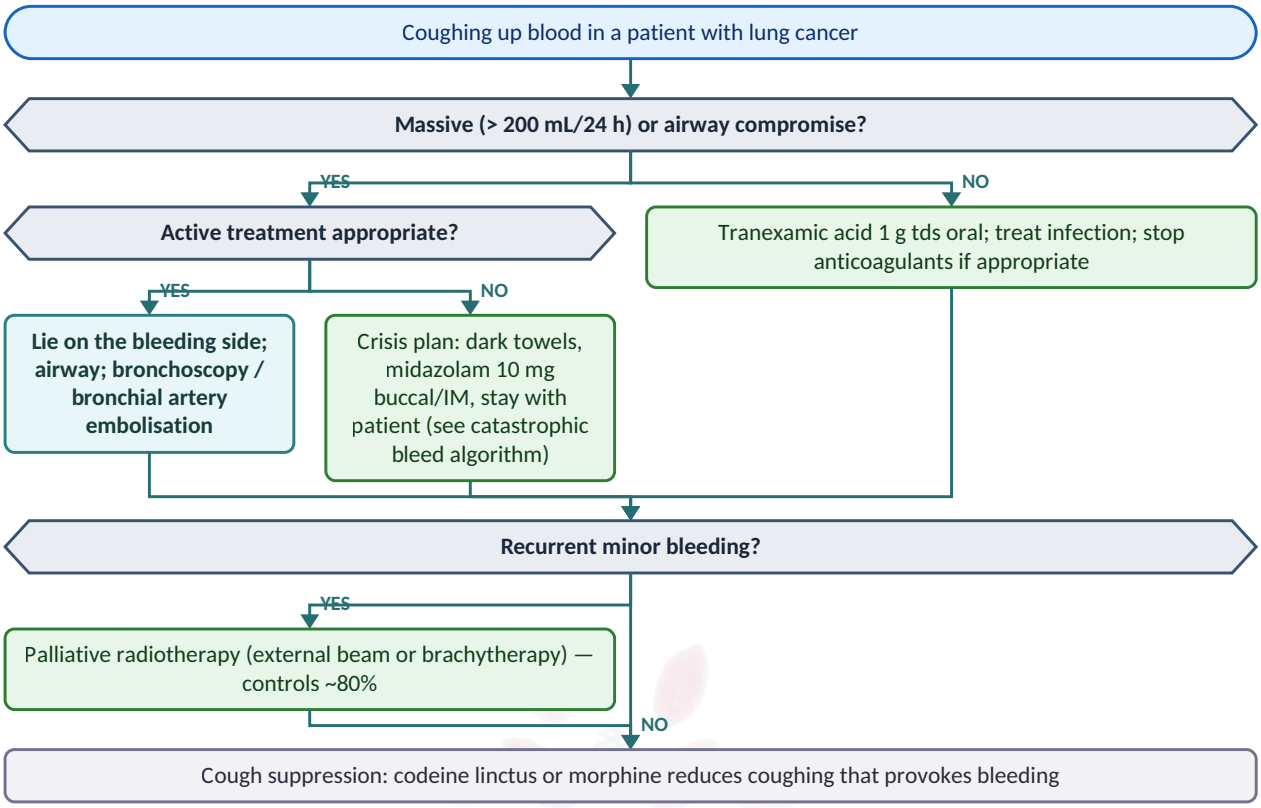
ALGORITHM ATLAS · Algo 58

Algorithm: Haemoptysis

Read the text ›

From lung tumours or infection; massive haemoptysis is life-threatening.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Squamous cell tumours near the hilum carry the highest risk.
- Prepare families in advance where a terminal bleed is possible.

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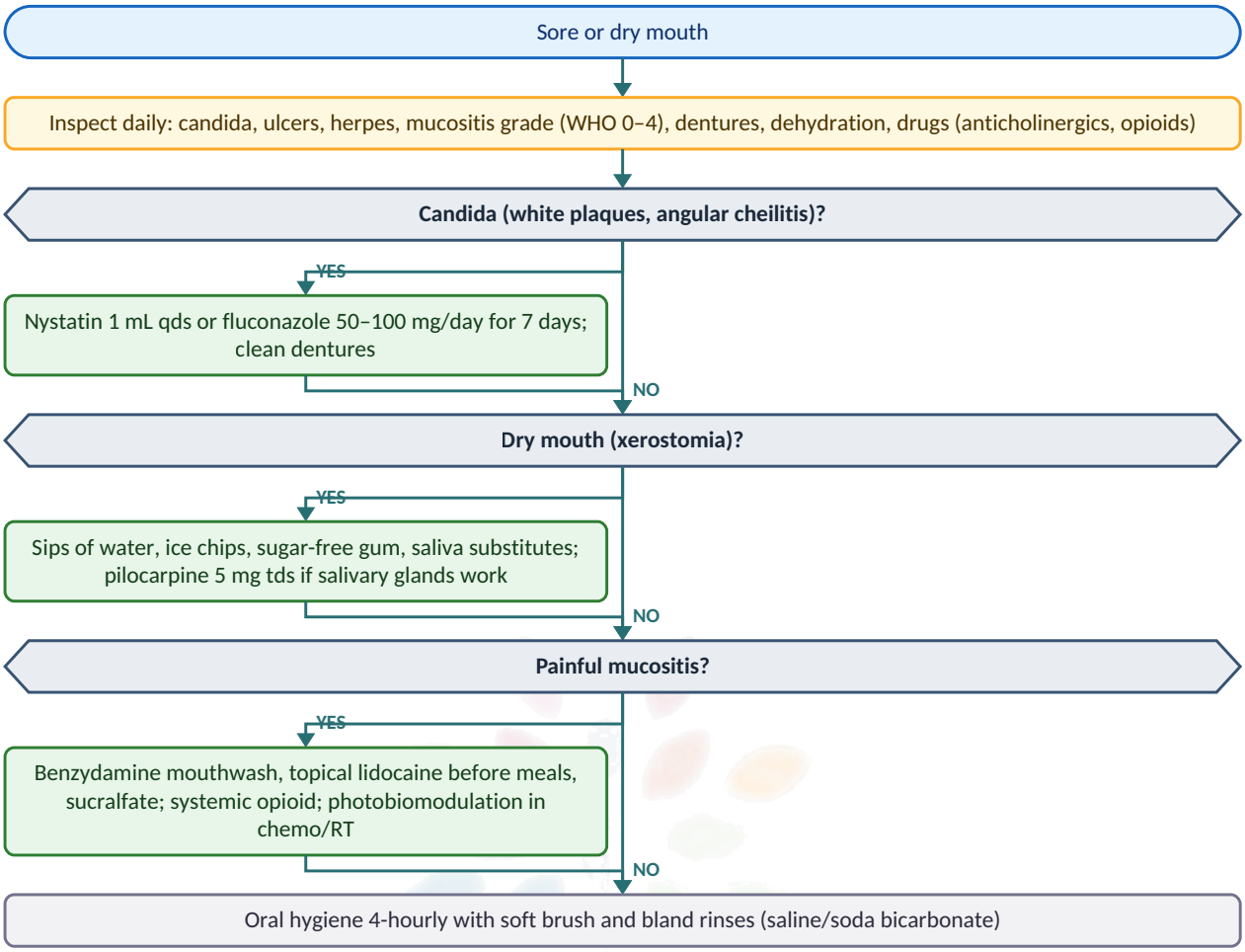
ALGORITHM ATLAS · Algo 59

Algorithm: Dry Mouth and Oral Mucositis

Read the text ›

Mouth problems impair eating, speaking and dignity.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- In dying patients, frequent mouth care relieves thirst better than IV fluids.
- Avoid lemon-glycerine swabs — they dry the mouth further.

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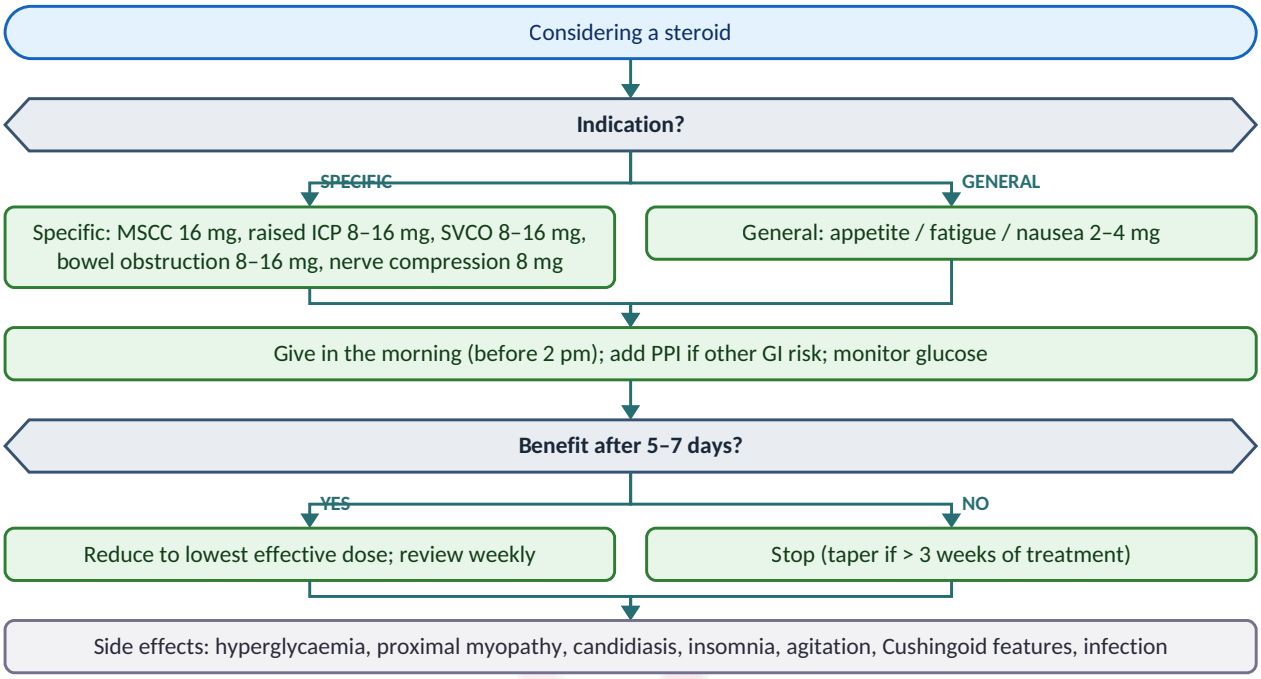
ALGORITHM ATLAS · Algo 60

Algorithm: Use of Corticosteroids in Palliative Care

Read the text ›

Dexamethasone is the most used steroid — powerful but with many side effects.

Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Proximal myopathy appears after 2–4 weeks and limits mobility.
- Steroid-induced hyperglycaemia peaks in the afternoon — check evening glucose.

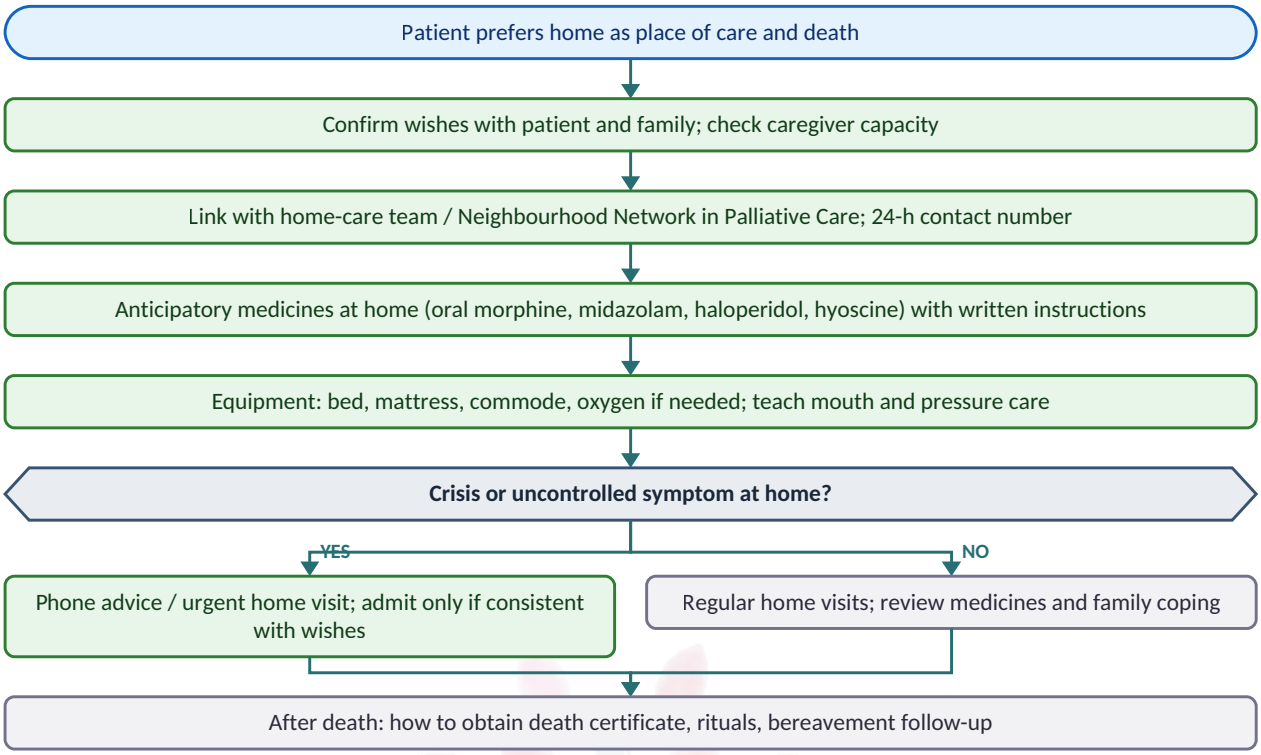
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ALGORITHM ATLAS · Algo 61

Algorithm: Planning for Death at Home

Most Indian patients wish to die at home; good planning avoids crisis admissions.

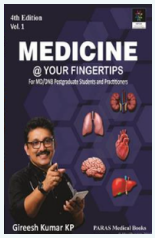
Start | Assess | Decision | Action | Urgent | Info | Outcome



★ KEY POINTS

- Kerala's community-owned model (NNPC) covers a large share of patients at home through volunteers.
- Narcotic Drugs and Psychotropic Substances Amendment Act 2014 eased morphine access.

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SOLVED CLINICAL CASE · Case 1

Cancer Pain with Chronic Kidney Disease

Read the text ›

A 62-year-old man with metastatic prostate cancer and stage 4 CKD (eGFR 22 mL/min) is on oral morphine 10 mg 4th-hourly for bone pain, with increasing drowsiness and myoclonic jerks over the last two days. Pain control itself has been adequate.

What is the most likely explanation for the new drowsiness and myoclonus?

Accumulation of morphine's active renally-excreted metabolites (morphine-6-glucuronide and morphine-3-glucuronide) due to reduced renal clearance, producing opioid neurotoxicity (sedation, myoclonus, and risk of respiratory depression) despite stable or even adequate analgesia.

What is the immediate management?

Reduce or temporarily withhold the morphine dose, ensure adequate hydration, and monitor sedation score and respiratory rate closely; treat myoclonus symptomatically if distressing (e.g. clonazepam) while switching opioid.

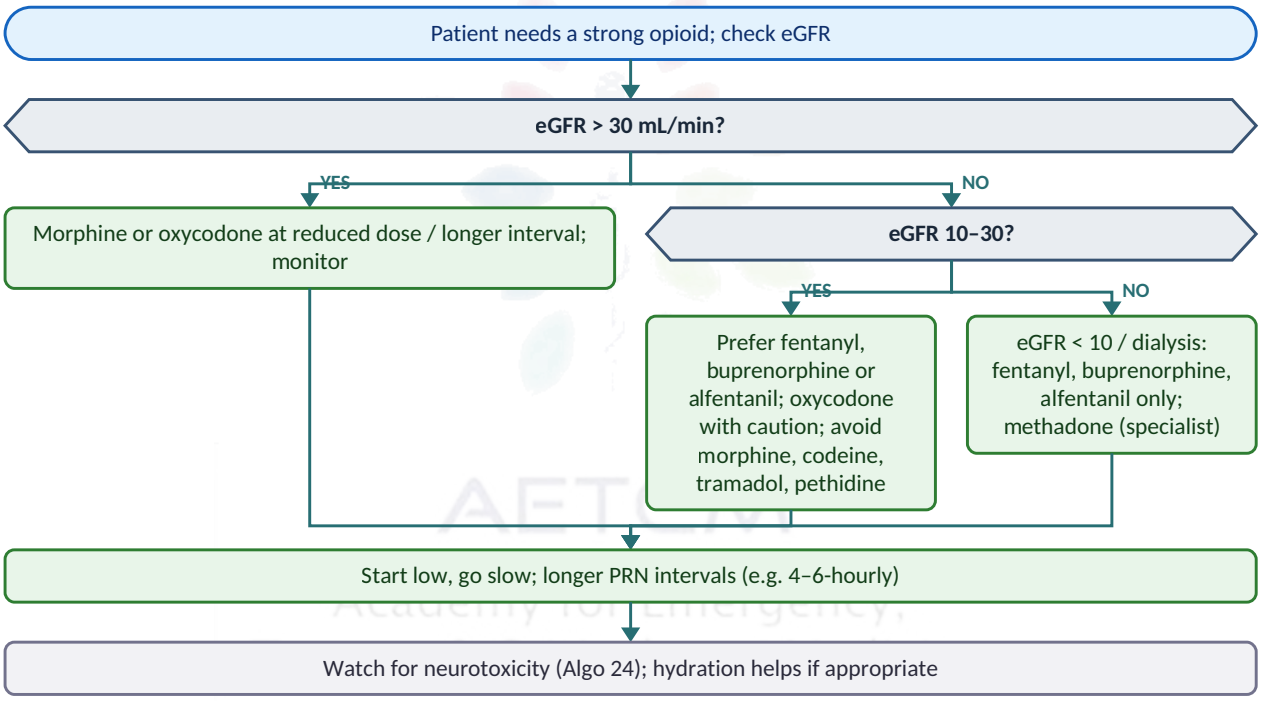
Which opioids are preferred once the patient is restabilised, and why?

Fentanyl or buprenorphine — both lack clinically significant active renal-excreted metabolites, making them safer choices in severe renal impairment; methadone is an alternative in specialist hands.

What dose adjustment principle applies when switching opioids here?

Calculate the equianalgesic dose from the current regimen, then reduce by at least 25–50% for incomplete cross-tolerance, titrating cautiously with close monitoring given the renal impairment.

ALGORITHM · Choosing an Opioid in Renal Impairment Algo 27 — full page ›



★ KEY POINTS

- Fentanyl and alfentanil are hepatically metabolised to inactive metabolites.
- Hydromorphone is intermediate — use with caution.

SOLVED CLINICAL CASE · Case 2

Breakthrough Pain on a Stable Opioid Regimen

Read the text ›

A 55-year-old woman with locally advanced breast cancer is on sustained-release morphine 30 mg 12th-hourly (60 mg/day) for bone and chest-wall pain, well controlled most of the day, but reports 3–4 episodes daily of sudden severe pain lasting 20–30 minutes, often when turning in bed.

How would you classify this pain, and what is the appropriate rescue dose?

This is incident (movement-related) breakthrough pain superimposed on well-controlled background pain. The rescue dose should be an immediate-release opioid at 1/6 of the total 24-h dose (IAPC) — here 60 ÷ 6 = 10 mg of oral morphine IR, available as needed.

How should the rescue dose be timed relative to the trigger?

Since the trigger (turning in bed/movement) is predictable, the rescue dose should be given 20–30 minutes before the anticipated activity for pre-emptive coverage.

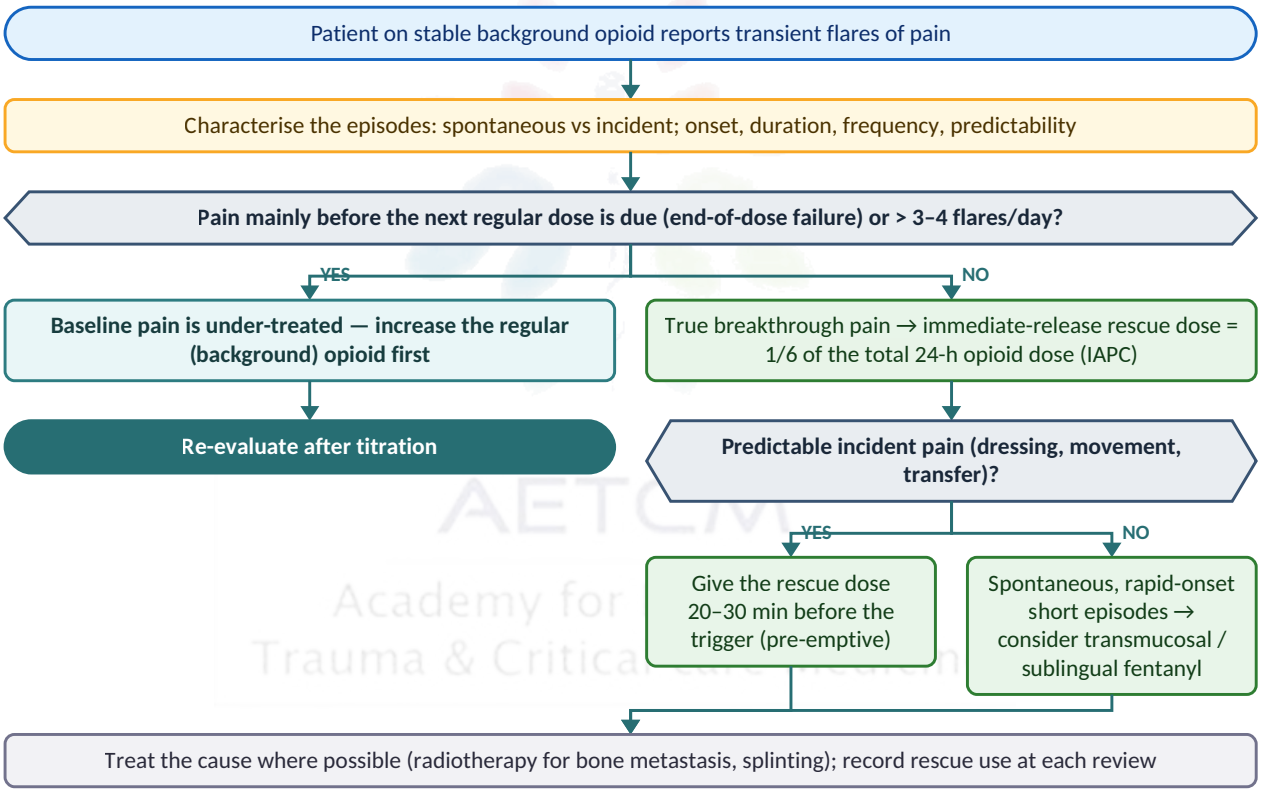
If episodes are very rapid in onset and short-lived, what alternative could be considered?

A rapid-onset, short-acting transmucosal or sublingual fentanyl preparation, which better matches the pharmacokinetic profile of rapid-onset incident pain compared with oral morphine.

Should the background dose be increased instead?

Not purely on the basis of predictable incident pain with well-controlled background pain; indiscriminately raising the background dose would risk excess sedation between episodes without addressing the trigger-related spikes.

ALGORITHM · Approach to Breakthrough Pain Algo 3 — full page ›



★ KEY POINTS

- Typical breakthrough episode peaks in 3–5 min and lasts < 30 min — oral morphine (onset 20–30 min) is often too slow.
- Transmucosal fentanyl doses are titrated independently, not as a % of background dose.

SOLVED CLINICAL CASE · Case 3

Post-Mastectomy Neuropathic Pain

A 48-year-old woman, six weeks after modified radical mastectomy and axillary clearance, describes a burning, electric-shock-like pain over the chest wall and inner arm, with allodynia on light touch. Opioids given so far have provided minimal relief.

What is the most likely diagnosis?

Post-mastectomy pain syndrome, a neuropathic pain condition related to intercostobrachial nerve injury during axillary surgery.

Why has the opioid trial been relatively ineffective?

Neuropathic pain responds poorly to opioids alone; first-line therapy for neuropathic pain is an adjuvant analgesic (anticonvulsant or antidepressant), not opioid escalation.

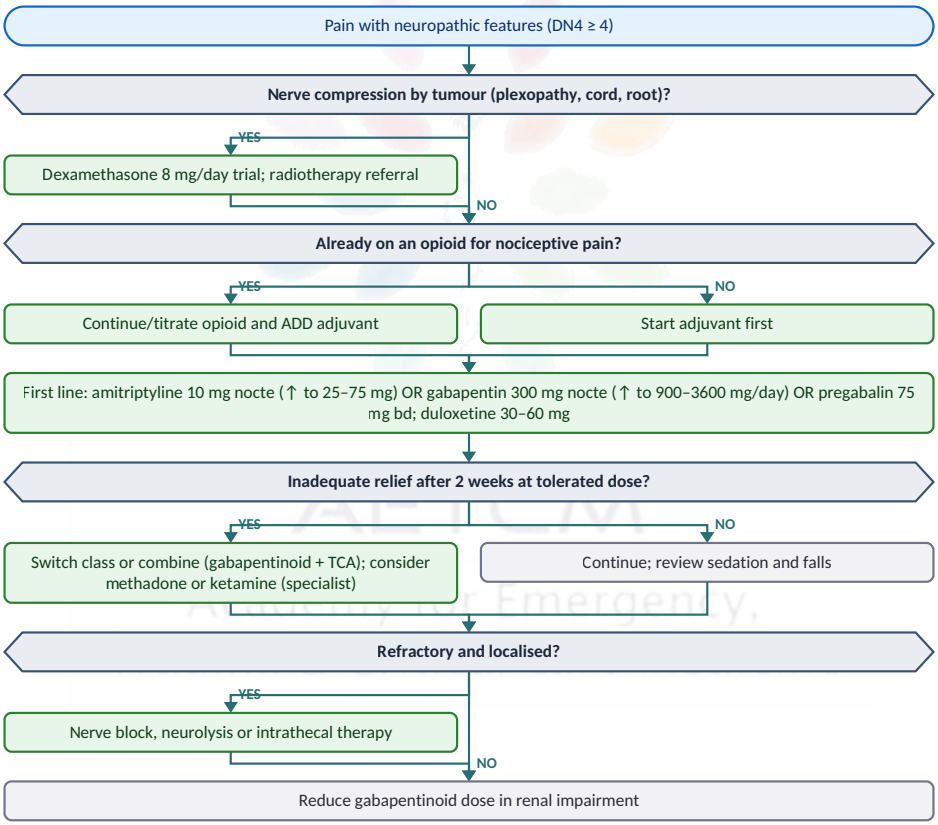
What first-line pharmacological options would you choose?

A gabapentinoid (gabapentin or pregabalin) or a tricyclic antidepressant (amitriptyline, started at low dose and titrated), either of which can be combined cautiously with a modest opioid dose if pain remains mixed/partially nociceptive.

What non-pharmacological measures are useful adjuncts?

Physiotherapy and desensitisation techniques, TENS, and psychological support (pain is often compounded by body-image and anxiety concerns after mastectomy).

ALGORITHM · Neuropathic Pain Pharmacotherapy Algo 25 — full page ›



★ KEY POINTS

- Number needed to treat ~3–4 for TCAs; ~6–8 for gabapentinoids.
- Amitriptyline: caution in cardiac disease and elderly.

SOLVED CLINICAL CASE · Case 4

Malignant Bowel Obstruction in Advanced Ovarian Cancer

Read the text ›

A 58-year-old woman with recurrent, inoperable ovarian cancer and peritoneal carcinomatosis presents with colicky abdominal pain, bilious vomiting, and absolute constipation for three days. She is cachectic with a poor performance status; the oncology team feels she is not a surgical candidate.

What is the diagnosis and how would you confirm it?

Inoperable malignant bowel obstruction; confirmed clinically and with a supine/erect abdominal X-ray or CT showing dilated bowel loops with air-fluid levels and a transition point.

What is the core medical management approach, given surgery is not appropriate?

Combination of an antisecretory agent (octreotide or hyoscine butylbromide) to reduce gastrointestinal secretions, an antiemetic (haloperidol preferred over prokinetics, which can worsen colic in complete obstruction), and analgesia (opioid, avoiding prokinetic-type adjuncts); a trial of dexamethasone may help resolve a partial/inflammatory component.

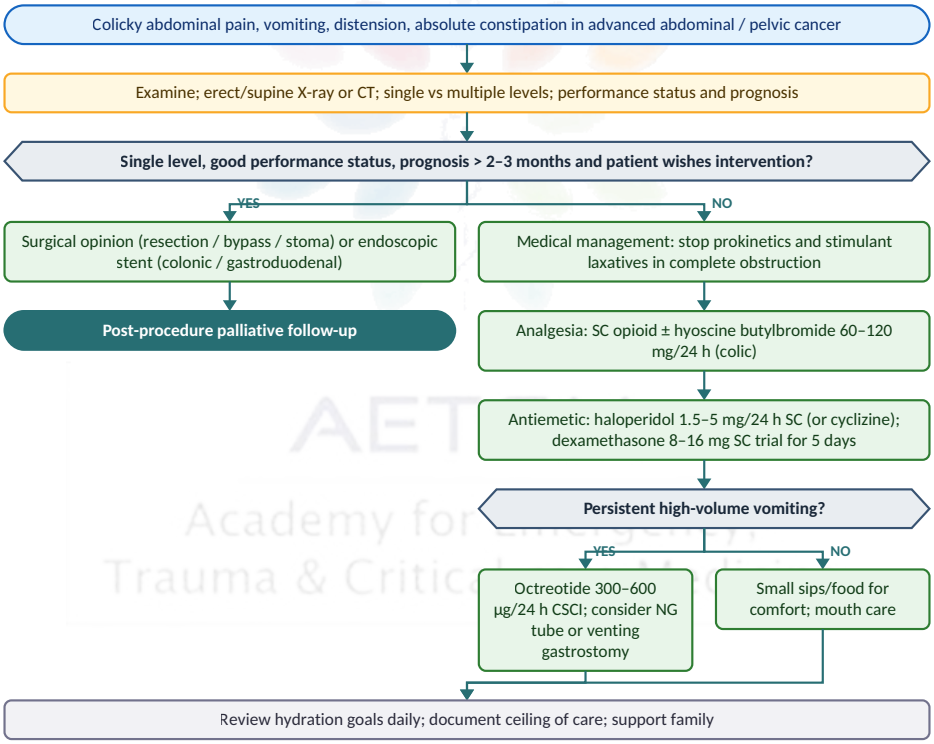
When is a nasogastric tube indicated?

Only for symptomatic relief when vomiting remains uncontrolled despite medical management, not as a routine or first-line measure.

What can be offered for ongoing oral intake?

Small volumes of food/fluid as tolerated for comfort and quality of life, accepting some vomiting may occur, rather than enforcing strict nil-by-mouth, unless this itself is distressing to the patient.

ALGORITHM · Approach to Malignant Bowel Obstruction Algo 4 — full page ›



★ KEY POINTS

- Metoclopramide is used only in partial (functional) obstruction without colic.
- Venting PEG frees the patient from a nasogastric tube in refractory obstruction.

SOLVED CLINICAL CASE · Case 5

Breathlessness in Advanced Lung Cancer

A 67-year-old man with metastatic non-small-cell lung cancer reports progressively worsening breathlessness at rest over two weeks. Oxygen saturation is 94% on room air. Chest X-ray shows a moderate pleural effusion and diffuse lymphangitic spread.

What are the key reversible contributors to assess for first?

Pleural effusion (potentially treatable by drainage), pulmonary embolism, anaemia, infection, and bronchospasm — each should be specifically considered before labelling breathlessness as purely due to disease progression.

How would you manage the pleural effusion component?

Therapeutic thoracocentesis for symptomatic relief; if effusion recurs and prognosis/performance status permit, consider an indwelling pleural catheter or pleurodesis.

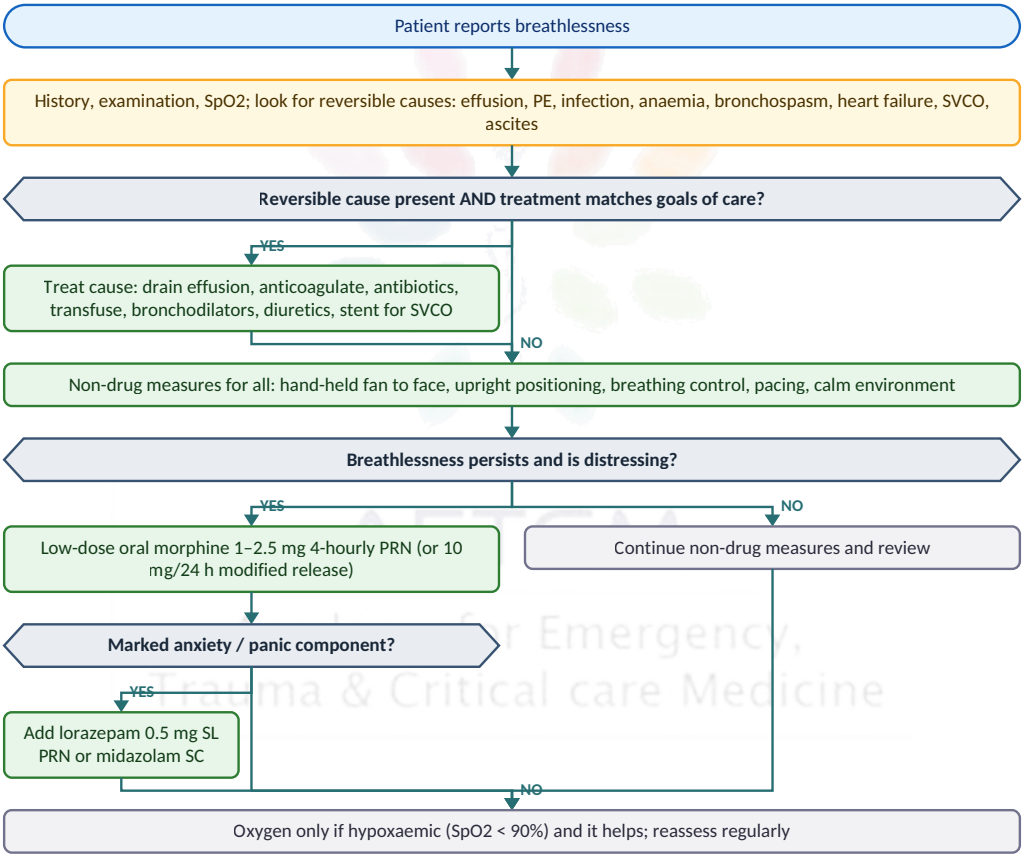
Is oxygen indicated here given the saturation of 94%?

Oxygen should be used based on symptomatic benefit and hypoxia, not reflexively; a trial can be given and continued only if it demonstrably improves breathlessness, since benefit is not guaranteed even with mild hypoxia.

What pharmacological option helps breathlessness once reversible causes are addressed/excluded?

Low-dose oral or parenteral opioids are first-line for symptomatic relief of refractory dyspnoea; a benzodiazepine may be added if anxiety is a significant component.

ALGORITHM · Approach to Breathlessness (Dyspnoea) in Palliative Care Algo 5 — full page ›



★ KEY POINTS

- Opioids reduce the sensation of breathlessness without clinically significant respiratory depression at low doses.
- The fan directed at the face (trigeminal stimulation) is evidence-based and costless.

SOLVED CLINICAL CASE · Case 6

Terminal Delirium in the Last Days of Life

Read the text ›

A 71-year-old woman with end-stage pancreatic cancer, now in the dying phase, becomes acutely agitated, picking at her sheets and calling out, distressing her family at the bedside.

What is the first step in management?

Screen for readily reversible and treatable contributors — urinary retention, faecal impaction, uncontrolled pain, opioid toxicity, and hypoxia — while recognising that in this terminal context extensive investigation is not appropriate.

If no easily reversible cause is found or correction is not appropriate, what is first-line pharmacological management?

Haloperidol, as the first-line antipsychotic for distressing terminal agitation/delirium; levomepromazine or olanzapine are alternatives.

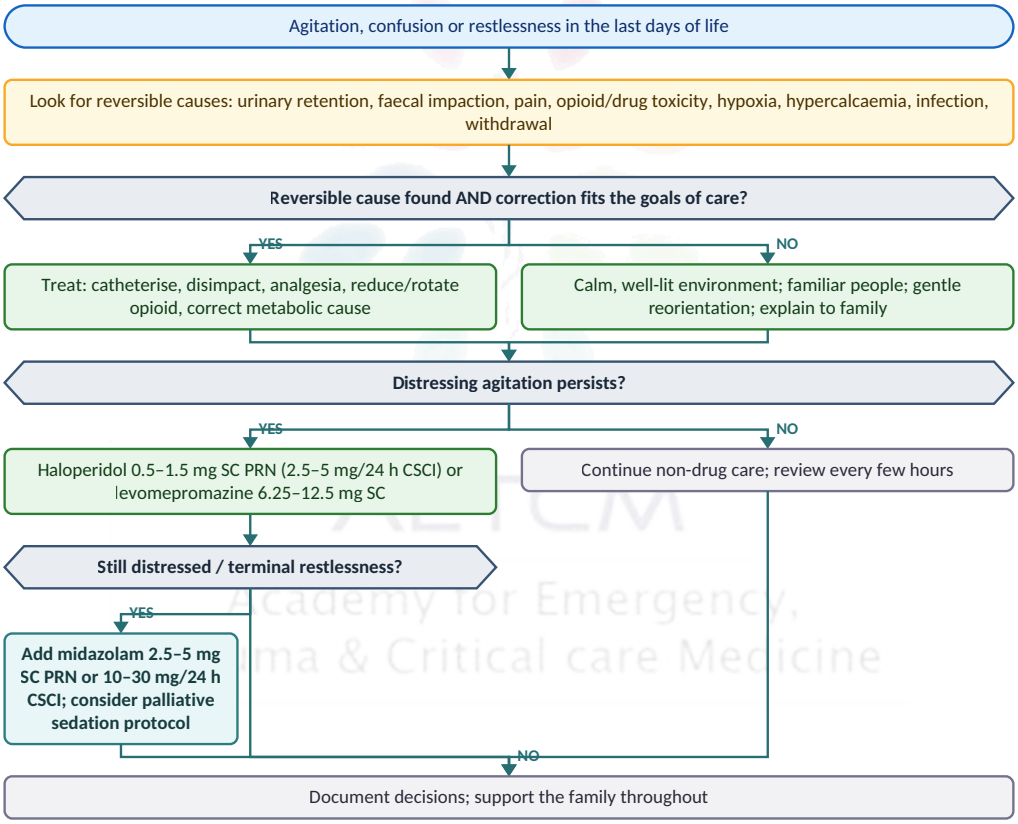
Why should benzodiazepines not be used alone as first-line here?

Benzodiazepines alone can worsen or precipitate delirium, particularly the hypoactive type, and are reserved as an add-on for persistent agitation or where sedation itself is the goal in terminal restlessness.

How should the family be supported through this?

Explain that terminal agitation is a recognised and treatable part of the dying process, reassure them it is not usually a sign of pain or distress once treated, and involve them gently in care and presence at the bedside.

ALGORITHM · Approach to Terminal Delirium / Agitation Algo 6 — full page ›



★ KEY POINTS

- Benzodiazepine alone can worsen delirium — give it with, not instead of, an antipsychotic (unless alcohol withdrawal).
- Hypoactive delirium is common and often missed.

SOLVED CLINICAL CASE · Case 7

Suspected Spinal Cord Compression

Read the text ›

A 59-year-old man with known multiple myeloma develops rapidly progressive bilateral leg weakness and urinary retention over 24 hours, with a band-like mid-back pain.

What is the most urgent diagnosis to exclude?

Malignant spinal cord compression — an oncological emergency requiring immediate action to prevent permanent paraplegia.

What is the immediate first step, even before imaging is obtained?

Start high-dose corticosteroids (e.g. dexamethasone) immediately with gastroprotection, and arrange urgent whole-spine MRI within 24 hours; do not delay steroids awaiting imaging.

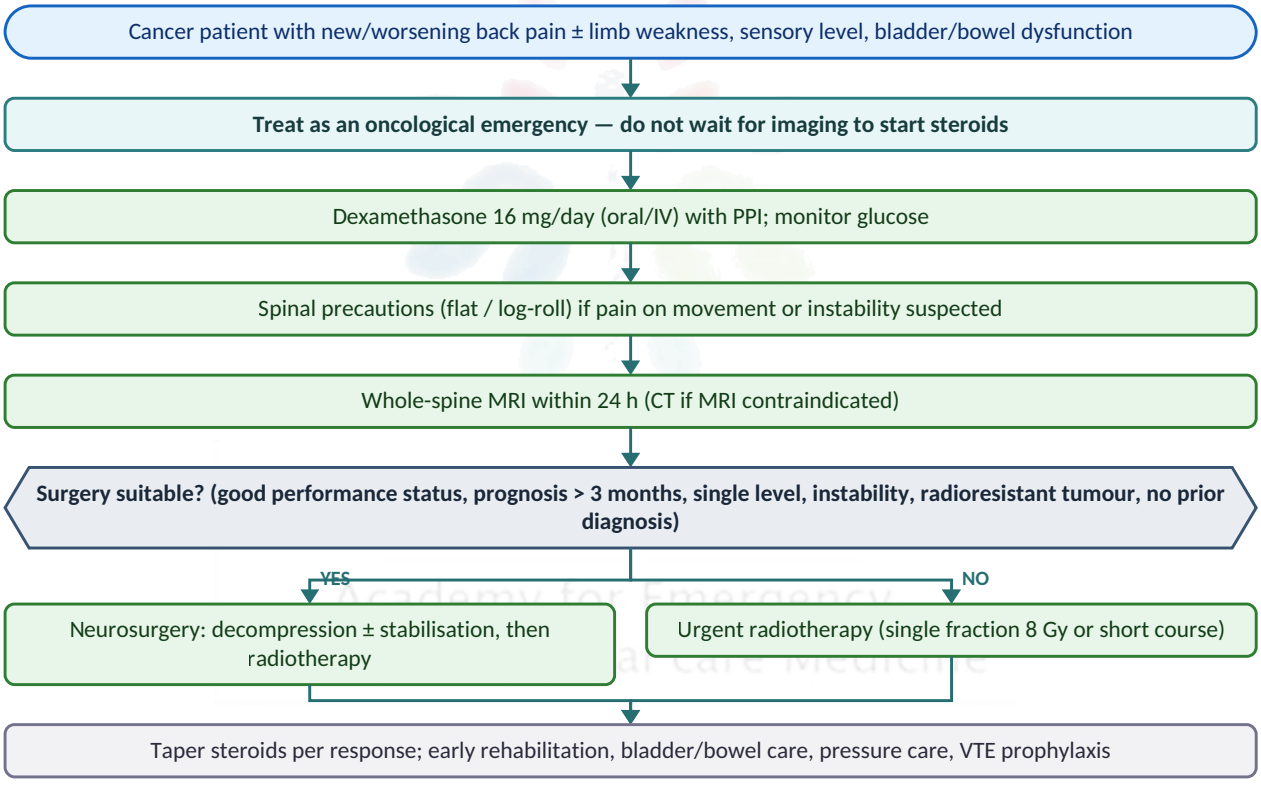
What determines whether surgery or radiotherapy alone is chosen?

Performance status, single vs multiple levels of compression, spinal instability, and radiosensitivity of the tumour; surgical decompression (with or without stabilisation) is favoured when feasible, followed by radiotherapy, while radiotherapy alone is used when surgery is not appropriate.

What is the prognosis for neurological recovery?

Strongly dependent on the degree and duration of weakness before treatment — patients who are still ambulant at diagnosis have the best chance of retaining mobility; established paraplegia for more than 24–48 hours rarely recovers fully, underscoring the need for urgent recognition and treatment.

ALGORITHM · Approach to Suspected Malignant Spinal Cord Compression Algo 9 — full page ›



★ KEY POINTS

- Ambulatory status at diagnosis is the strongest predictor of outcome.
- Patchell trial (2005): surgery + RT kept more patients walking than RT alone in selected cases.

SOLVED CLINICAL CASE · Case 8

Hypercalcaemia of Malignancy

Read the text ›

A 64-year-old woman with metastatic breast cancer presents with confusion, severe constipation, and polyuria. Corrected serum calcium is 13.8 mg/dL.

What is the diagnosis and immediate priority?

Severe symptomatic hypercalcaemia of malignancy; immediate priority is aggressive rehydration with IV isotonic saline, as most patients are significantly volume-depleted.

What definitive pharmacological treatment follows rehydration?

An intravenous bisphosphonate (e.g. zoledronic acid) once the patient is adequately rehydrated; this takes 2–4 days for full effect.

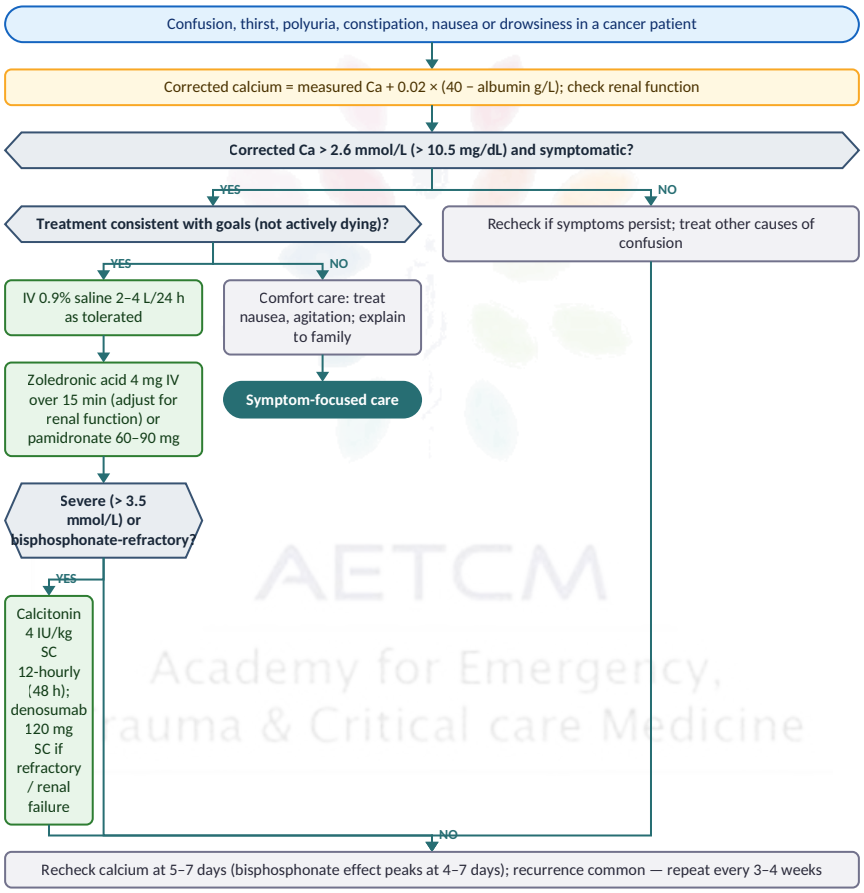
What can be used for more rapid symptom control while awaiting bisphosphonate effect?

Calcitonin, which has a rapid but short-lived calcium-lowering effect, useful as a bridge in severely symptomatic patients.

What should guide the decision to correct hypercalcaemia versus manage symptomatically only?

The patient's overall prognosis and goals of care — in a patient actively dying with days to live, aggressive correction may not be appropriate, and comfort-focused symptomatic management is preferred instead.

ALGORITHM · Approach to Hypercalcaemia of Malignancy Algo 10 — full page ›



★ KEY POINTS

- Median survival after cancer hypercalcaemia is often weeks to months — discuss goals.
- PTHrP-mediated humoral hypercalcaemia is commonest (squamous carcinomas).

SOLVED CLINICAL CASE · Case 9

Opioid Toxicity

Read the text ›

A 70-year-old man on oral morphine for cancer pain, recently increased for poorly controlled pain, is found markedly drowsy with a respiratory rate of 8/min and pinpoint pupils.

What is the diagnosis?

Opioid-induced respiratory depression/toxicity, likely from an excessive or too-rapid dose increase.

What is the immediate management?

Stop/withhold the opioid, support airway and breathing, and if respiratory depression is significant, give titrated naloxone (0.04–0.1 mg IV boluses) to reverse toxicity while minimising precipitation of acute pain crisis and withdrawal.

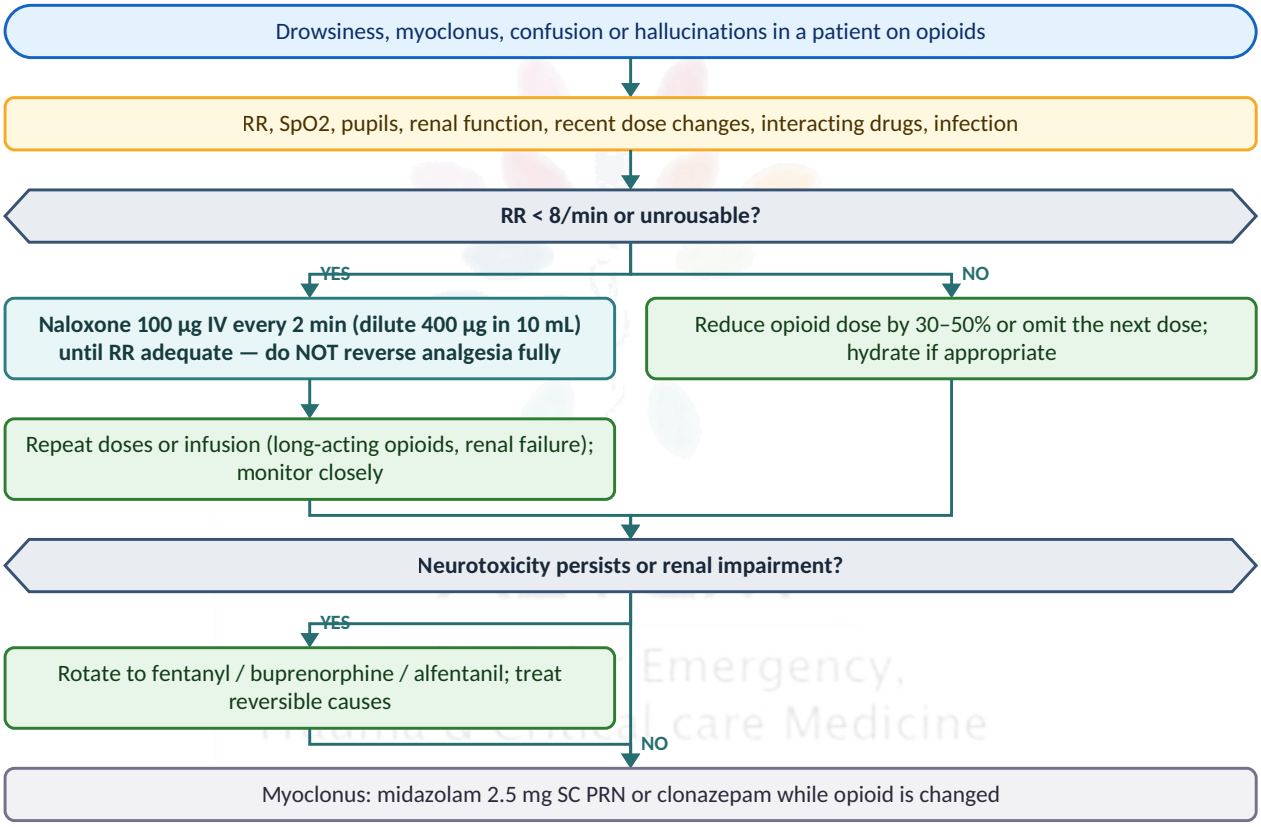
Why is cautious, titrated naloxone dosing preferred over a single large dose?

A large naloxone bolus can precipitate acute, severe pain and an acute withdrawal reaction in an opioid-tolerant patient; titrated small boluses aim to restore adequate ventilation while preserving some analgesia.

What monitoring is needed after naloxone administration?

Close observation for re-sedation, since naloxone's half-life is shorter than that of morphine (and much shorter than long-acting opioids), often requiring repeat doses or an infusion.

ALGORITHM · Opioid Toxicity and Opioid-Induced Neurotoxicity Algo 24 — full page ›



★ KEY POINTS

- Full-dose naloxone in opioid-tolerant patients causes severe pain and withdrawal.
- Morphine-3- and -6-glucuronide accumulate in renal failure.

SOLVED CLINICAL CASE · Case 10

Total Pain in Advanced Cancer

Read the text ›

A 52-year-old man with metastatic colorectal cancer describes pain as '10/10, nothing works,' despite being on an adequately titrated strong opioid regimen with good adherence. He appears withdrawn, admits to significant financial strain from treatment costs, and voices fear about what happens to his family after he dies.

What concept best explains this scenario?

Cicely Saunders' concept of 'total pain' — physical pain amplified and compounded by unresolved psychological distress, social/financial strain, and spiritual/existential concerns, which analgesic escalation alone cannot address.

What is the risk of simply escalating the opioid dose further?

Increased dose without addressing the non-physical contributors risks opioid toxicity and adverse effects without proportionate benefit, since the physical component is only part of the overall suffering.

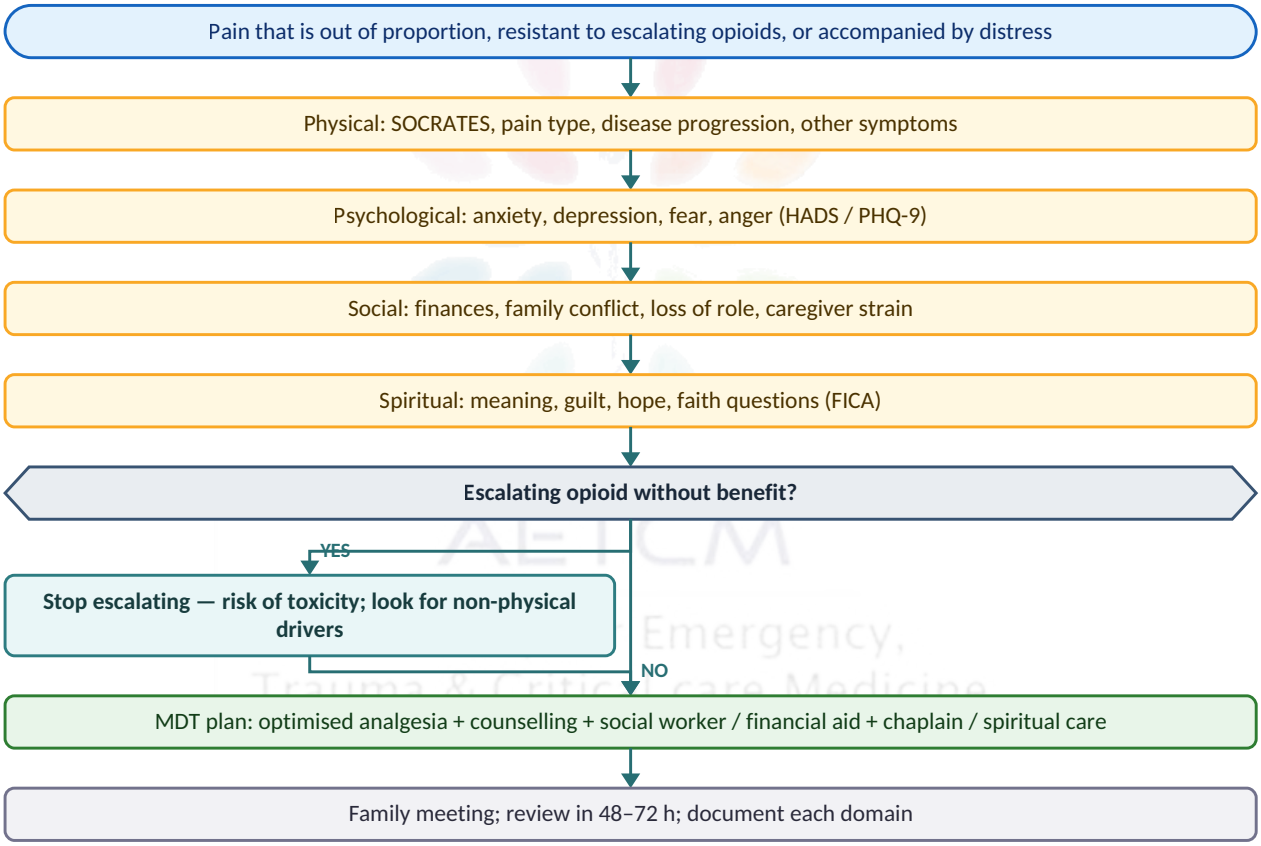
What multidisciplinary approach is indicated?

Involvement of psychological counselling/psychiatry for distress, medical social work for financial and family support, and spiritual/chaplaincy support, alongside continued optimisation of physical symptom control.

How does this case illustrate the role of the palliative care team structure?

It demonstrates the need for a multidisciplinary team (physician, nurse, counsellor, social worker, chaplain/ spiritual care provider) working together, rather than a purely pharmacological, physician-only response.

ALGORITHM · Assessment and Management of Total Pain Algo 31 — full page ›



★ KEY POINTS

- Chemical coping (using opioids for emotional distress) signals unaddressed total pain.
- Ask: 'What worries you most?' — often reveals the hidden domain.

SOLVED CLINICAL CASE · Case 11

Pediatric Palliative Pain

A 9-year-old boy with relapsed osteosarcoma and femoral metastases has severe limb pain, inadequately controlled on paracetamol and a weak opioid.

What step of the WHO approach to pediatric pain management is indicated next?

Step up to a strong opioid (e.g. oral morphine), as the WHO 2-step strategy for children recommends moving directly from non-opioids to strong opioids for moderate-to-severe pain, rather than persisting with weak opioids.

What special considerations apply to opioid dosing in children?

Weight-based dosing with careful titration, use of age-appropriate formulations, and involvement of caregivers in administration and monitoring; starting doses are lower and adjusted to body weight compared with adults.

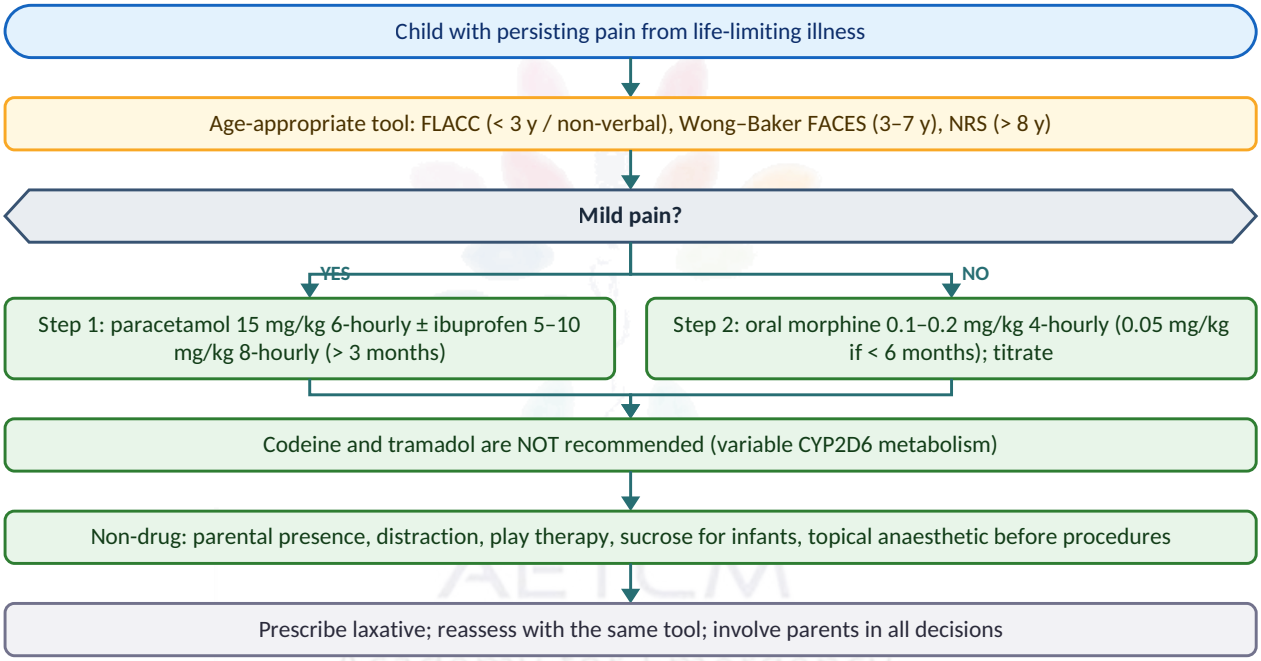
What non-pharmacological measures are especially important in pediatric palliative pain?

Play therapy, distraction techniques, parental presence and comfort, and age-appropriate explanation of procedures and pain, alongside pharmacological treatment.

How should pain be assessed in this age group?

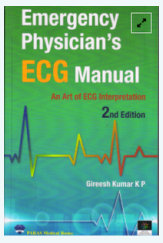
Using validated self-report tools appropriate to age and cognitive ability (e.g. Wong-Baker FACES scale, numerical rating scale in older children) supplemented by behavioural observation.

ALGORITHM · Pain Management in Children Algo 32 — full page ›



★ KEY POINTS

- Children are not small adults — dose by weight and age.
- ACT categories 1–4 classify life-limiting conditions for paediatric palliative care.



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SOLVED CLINICAL CASE · Case 12

Non-Cancer Palliative Care — End-Stage Renal Disease

A 78-year-old man with end-stage renal disease has chosen conservative kidney management (no dialysis) after discussion with his nephrologist and family. He is referred to the palliative care team for symptom management and advance care planning.

Is palliative care appropriate here, given this is a non-malignant condition?

Yes — palliative care is appropriate for any life-limiting illness, not only cancer, and conservative (non-dialysis) management of ESRD is a recognised indication for an integrated palliative approach.

What are common symptoms requiring attention in conservatively managed ESRD?

Pruritus, fatigue, nausea, pain (often requiring caution with renally-cleared drugs), fluid overload symptoms, and uraemic symptoms as disease progresses.

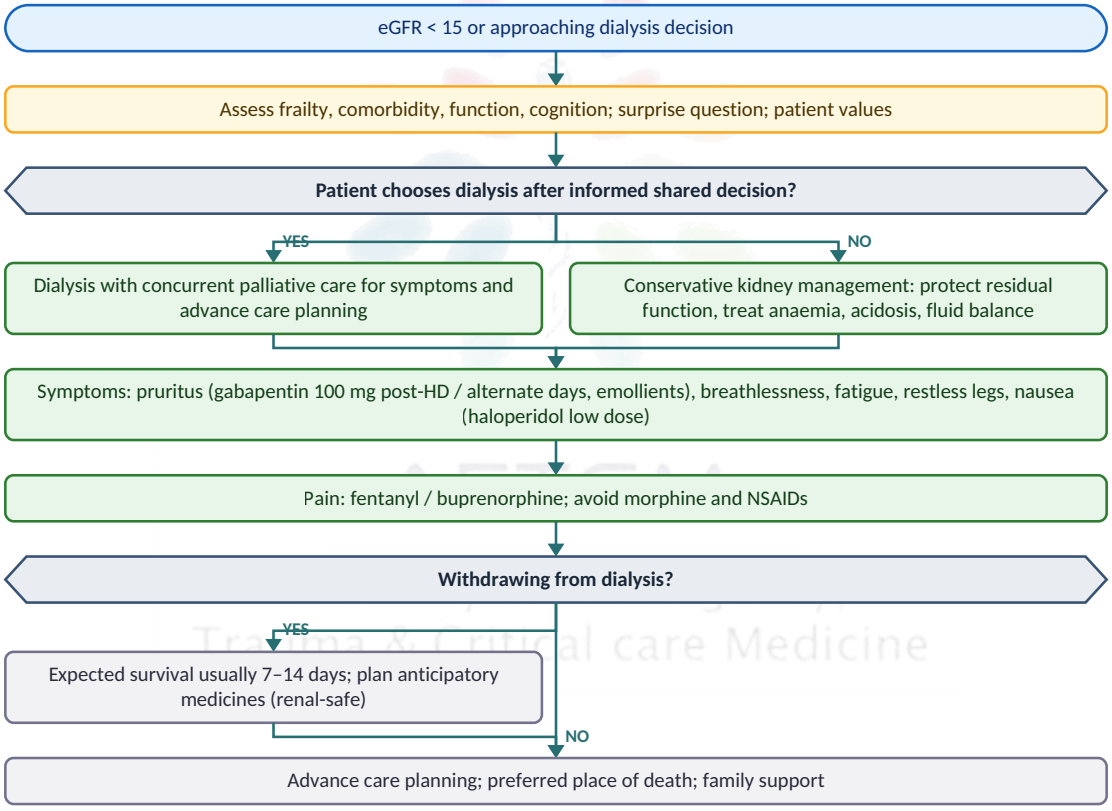
What opioid considerations apply, given his renal function?

Preference for opioids without active renally-excreted metabolites (fentanyl, buprenorphine) with cautious low starting doses and careful titration.

What else should the palliative care team address proactively?

Advance care planning and discussion of preferences for future deterioration, psychosocial support for the patient and family coping with a non-dialysis pathway, and anticipatory prescribing as the terminal phase approaches.

ALGORITHM · Conservative Kidney Management (End-Stage Kidney Disease) Algo 33 — full page ›



★ KEY POINTS

- Over-80s with high comorbidity may survive as long on conservative management as on dialysis.
- Midazolam and alfentanil are preferred in the dying renal patient.

SOLVED CLINICAL CASE · Case 13

End-Stage COPD with Refractory Breathlessness

A 74-year-old man with end-stage COPD (on home oxygen) is admitted with severe breathlessness at rest despite optimised inhaled bronchodilator therapy and no evidence of infection or pneumothorax.

What is the next step in symptomatic management, given reversible causes have been excluded?

A trial of low-dose oral opioid (e.g. low-dose morphine) for refractory breathlessness, which has evidence for symptomatic benefit in advanced COPD without a clinically significant increase in respiratory depression at these low doses when appropriately titrated.

What non-pharmacological measure has evidence for breathlessness relief?

A hand-held fan directed at the face, which stimulates trigeminal afferents and reduces the sensation of breathlessness.

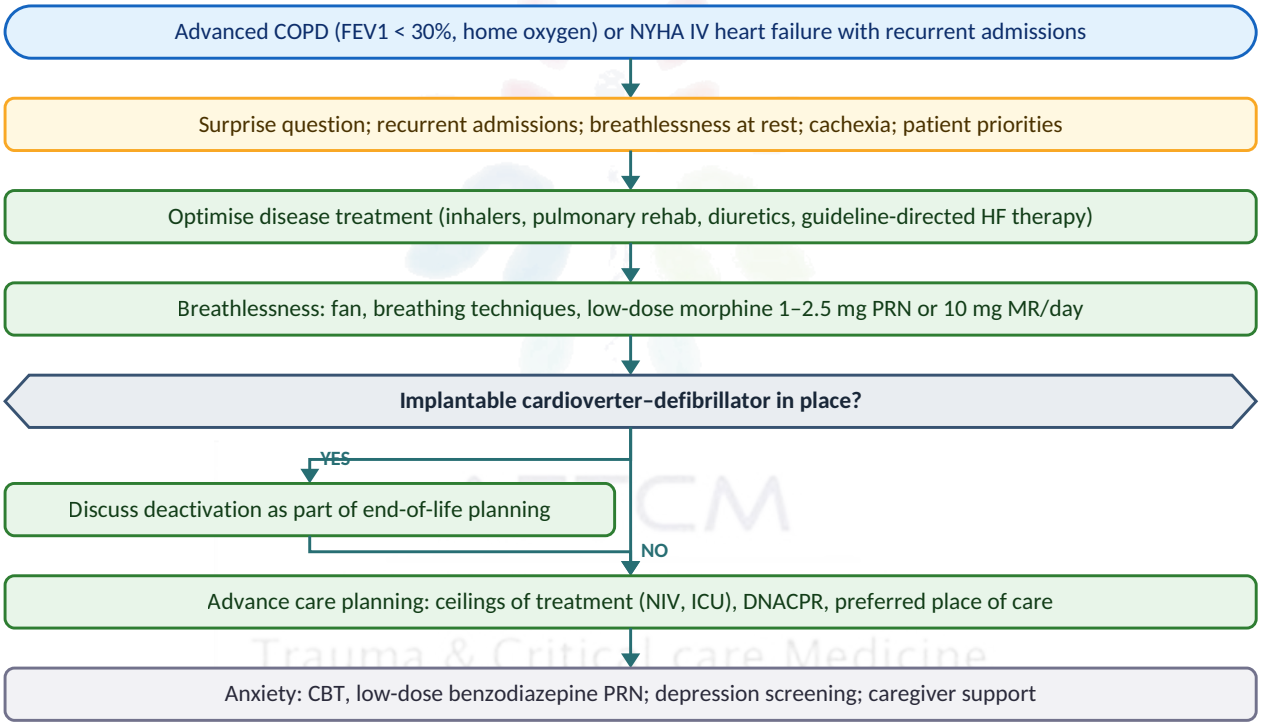
How does palliative care differ from, or complement, ongoing respiratory/disease-specific management here?

Palliative care runs in parallel with continued optimal COPD management (bronchodilators, home oxygen, pulmonary rehabilitation where feasible) rather than replacing it — illustrating the early-integration model of palliative care in non-malignant chronic disease.

What advance care planning discussion is relevant at this stage?

Discussing preferences regarding future hospitalisation, escalation of care (e.g. non-invasive ventilation, ICU), and resuscitation status, given the progressive, life-limiting nature of end-stage COPD.

ALGORITHM · End-Stage COPD and Heart Failure Algo 34 — full page ›



★ KEY POINTS

- Opioids for breathlessness in COPD do not cause clinically important hypercapnia at low doses.
- Prognosis is hard to predict — use triggers, not timelines.

SOLVED CLINICAL CASE · Case 14

Advanced Dementia — Feeding and End-of-Life Dilemma

> *An 86-year-old woman with advanced dementia has progressive dysphagia and recurrent aspiration pneumonia. The family asks whether a feeding tube should be placed to 'help her live longer.'*

What does the evidence say about feeding tubes in advanced dementia?

Tube feeding in advanced dementia has not been shown to prolong survival, prevent aspiration pneumonia, improve nutritional status, or improve quality of life compared with careful hand feeding, and carries its own risks and burdens.

How should this be communicated to the family?

Empathetically explain the evidence and reframe the goal from prolonging life via a tube to maximising comfort and dignity via careful hand feeding, oral care, and attention to the patient's expressed or best-interests wishes.

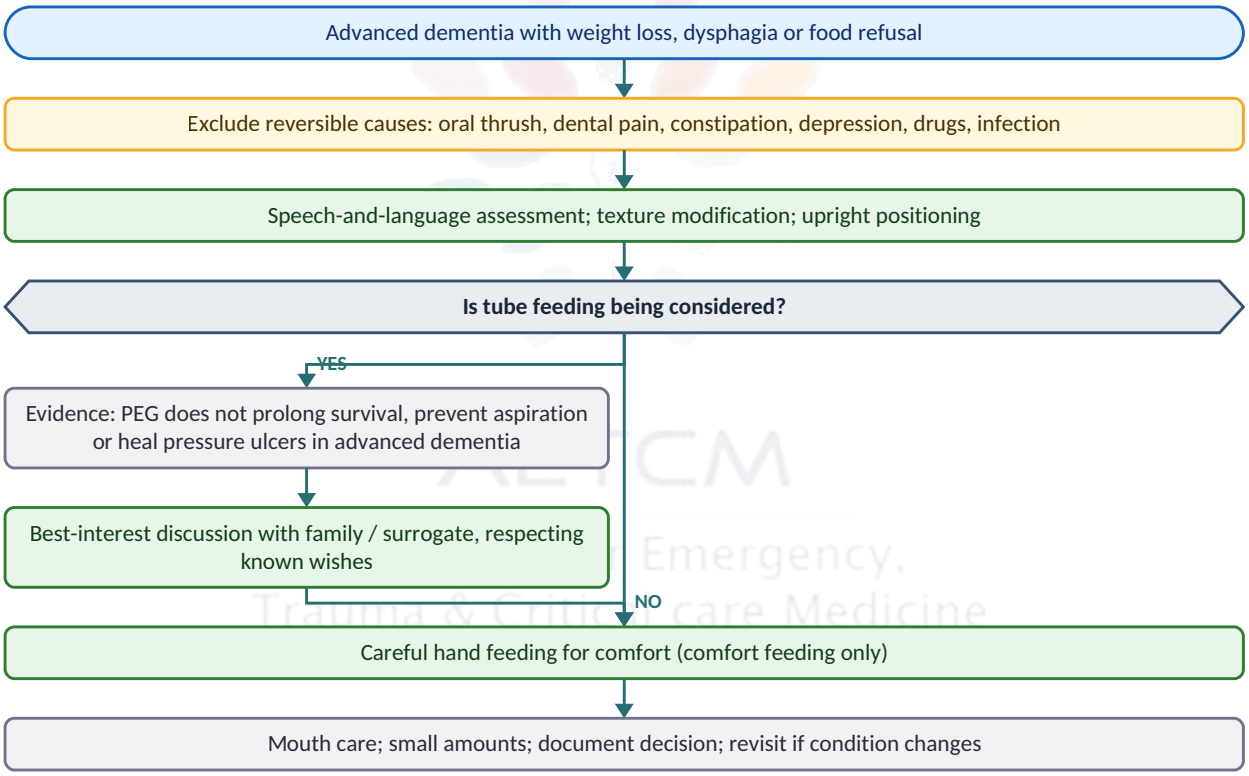
What ethical principles guide this decision?

Beneficence and non-maleficence (weighing burden versus benefit of the intervention), respect for patient autonomy (prior expressed wishes if known), and best-interests decision-making with the family as surrogate decision-makers.

What is the role of advance care planning in preventing this dilemma in future cases?

Early discussion of goals of care and documentation of preferences (including an Advance Medical Directive where applicable) before advanced dementia impairs decision-making capacity, so that crisis decisions are not left entirely to family at a late stage.

ALGORITHM · Feeding Decisions in Advanced Dementia Algo 35 — full page ›



★ KEY POINTS

- Hand feeding offers human contact that tube feeding removes.
- Aspiration pneumonia is common with PEG too (reflux of feeds).

MODEL LONG-ESSAY ANSWER · LE 1

The WHO Analgesic Ladder: Principles, Evidence and Modern Modifications

Read the text ›

Introduction. The World Health Organization's three-step analgesic ladder, introduced in 1986, remains the foundational framework for cancer pain management worldwide, achieving adequate relief in the large majority of patients when applied correctly.

The original three steps. Step 1 (mild pain, 1–3/10) uses a non-opioid — paracetamol or an NSAID — with or without an adjuvant. Step 2 (mild–moderate pain, 4–6/10) adds a weak opioid such as tramadol or codeine to the Step 1 regimen. Step 3 (moderate–severe pain, 7–10/10) substitutes a strong opioid — typically morphine — for the weak opioid, continuing non-opioids and adjuvants as indicated.

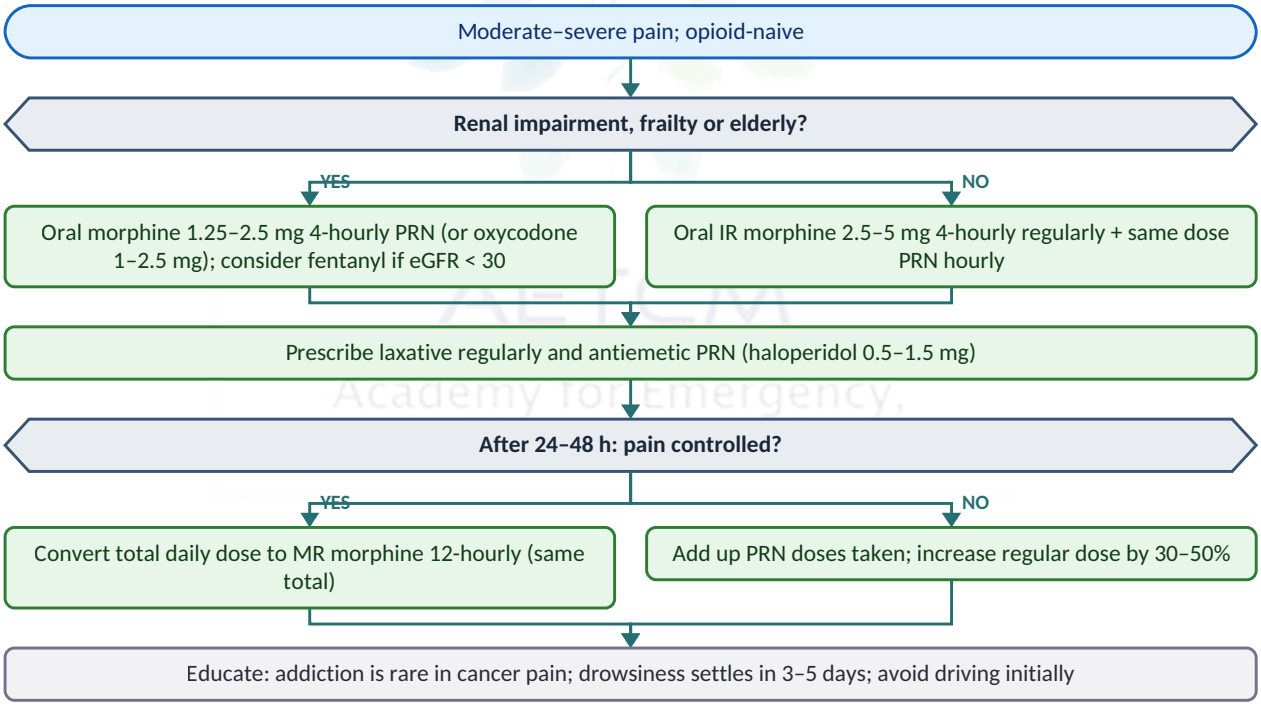
Core principles. Analgesics should be given "by the clock" (regularly, not only on demand), "by the ladder" (stepwise according to severity), "by the appropriate route" (oral whenever possible), and "by the individual," with attention to detail and regular reassessment — principles that remain as important as the ladder itself.

Modern modifications and criticisms. Many contemporary practitioners bypass Step 2 for moderate–severe pain, moving directly to a low-dose strong opioid ("Step 2 skip"), since weak opioids have a ceiling effect and tramadol/codeine offer little advantage over low-dose morphine with fewer drug interactions. A proposed "fourth step" incorporates interventional techniques (nerve blocks, neuraxial analgesia, neurolytic procedures) for refractory pain unresponsive to the first three steps. The ladder has also been criticised for being insufficiently individualised, with some experts advocating a "two-step" or severity-based approach that selects the starting analgesic directly by pain intensity rather than rigid sequential stepping, reserving the ladder's conceptual structure (non-opioid → opioid → adjuvant) rather than its literal steps.

Adjuvant analgesics (antidepressants, anticonvulsants, corticosteroids, bisphosphonates, ketamine, muscle relaxants) may be added at any step, particularly for neuropathic or bone pain, rather than being confined to a specific rung.

Conclusion. The WHO ladder remains a globally applicable, resource-appropriate framework, particularly valuable in low-resource settings; its enduring strength lies less in the rigid three steps than in its underlying principles of regular, individualised, appropriately-routed analgesia with early adjuvant use.

ALGORITHM · Starting Opioids in an Opioid-Naive Patient Algo 29 — full page ›



MODEL LONG-ESSAY ANSWER · LE 2

Comprehensive Management of Pain due to Bone Metastases

Read the text ›

Introduction. Bone is among the commonest sites of metastasis in breast, prostate, lung, renal, and thyroid cancers, and bone pain is one of the most frequent and often most severe pain syndromes encountered in palliative medicine, requiring a multimodal approach.

Pathophysiology. Bone pain arises from a combination of nociceptive (periosteal stretch, microfracture, mechanical instability) and inflammatory mechanisms (osteoclast activation, prostaglandin and cytokine release), explaining why pain is often disproportionate to lesion size and responds well to anti-inflammatory and bone-targeted therapy, not opioids alone.

Pharmacological management. NSAIDs are particularly effective given the inflammatory component and are used at every WHO ladder step unless contraindicated. Opioids are titrated according to severity. Bisphosphonates (zoledronic acid) or denosumab (a RANK-ligand inhibitor) reduce skeletal-related events and have an analgesic effect, typically over 2–4 weeks; both require attention to renal function/calcium (bisphosphonates) and jaw health (risk of osteonecrosis of the jaw with either class). Corticosteroids provide rapid, if short-term, relief, particularly useful in acute flares or impending complications.

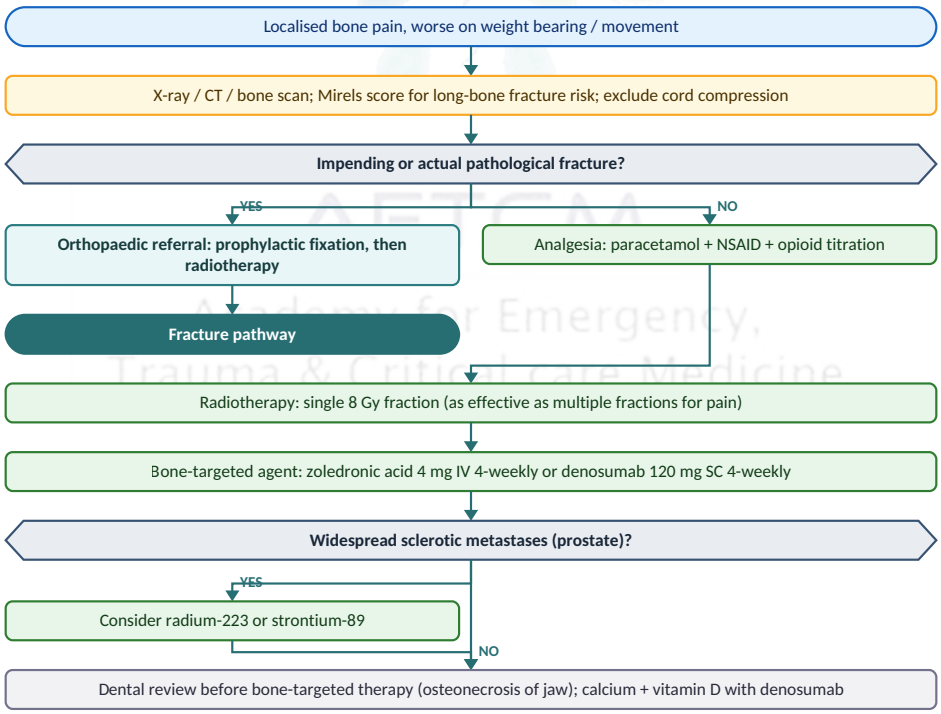
Radiotherapy. External beam radiotherapy is highly effective for localised bone pain, with a single 8 Gy fraction providing equivalent pain relief to multi-fraction regimens for uncomplicated bone metastases, an important resource-efficient option. For widespread painful bone metastases, radiopharmaceuticals (Strontium-89, Samarium-153) offer systemic pain relief, though with a lag to onset and bone-marrow suppression as the main toxicity.

Interventional and surgical options. Prophylactic fixation is considered for impending pathological fracture (e.g. Mirels' score-guided), vertebroplasty/kyphoplasty for vertebral compression, and targeted nerve blocks or neurolysis for refractory focal pain.

Non-pharmacological measures. Careful positioning, mobility aids, orthotic support, and physiotherapy reduce mechanical loading on weakened bone and contribute meaningfully to comfort.

Conclusion. Optimal bone pain management is multimodal — combining analgesics, bone-targeted agents, radiotherapy, and mechanical/surgical support — tailored to prognosis, fracture risk, and patient goals.

ALGORITHM · Pain from Bone Metastases Algo 26 — full page ›



MODEL LONG-ESSAY ANSWER · LE 3

Total Pain: Concept and Multidisciplinary Management

Read the text ›

Introduction. Dame Cicely Saunders, founder of the modern hospice movement, introduced the concept of "total pain" to capture the observation that suffering in advanced illness extends beyond nociception into psychological, social, and spiritual domains, each of which can amplify or be mistaken for physical pain.

The four domains. *Physical* — the nociceptive/neuropathic pain itself, plus co-existing symptoms (nausea, breathlessness, fatigue). *Psychological* — anxiety, depression, fear of dying, anger, and loss of control, all of which lower the pain threshold and amplify perceived pain intensity. *Social* — financial strain, loss of role (as breadwinner, parent, spouse), family conflict, and social isolation. *Spiritual* — loss of meaning, unresolved guilt, existential distress, and questions about what happens after death.

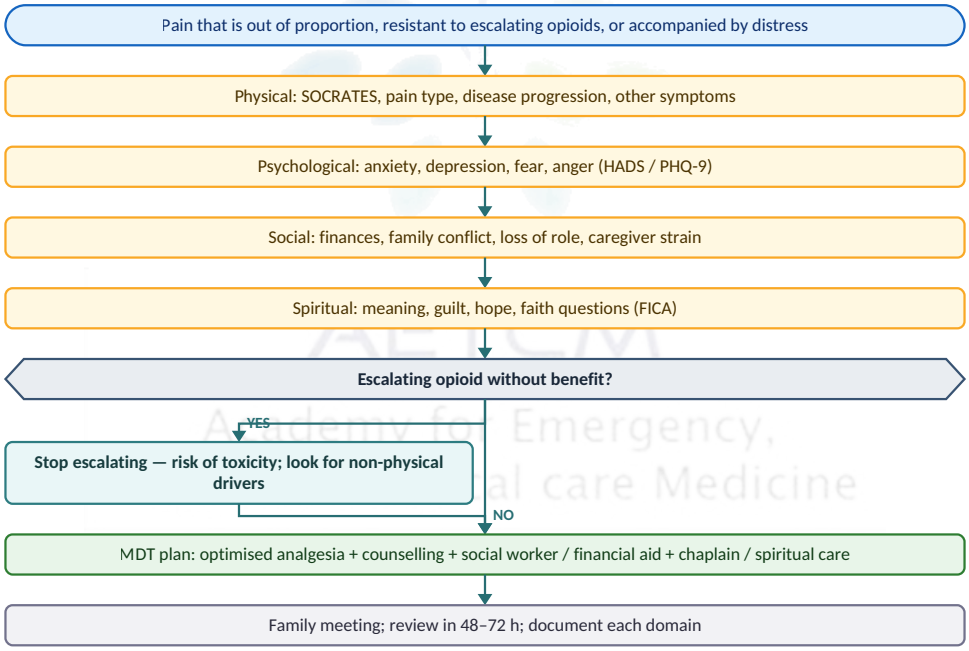
Clinical recognition. Total pain should be suspected when reported pain intensity is disproportionate to objective findings, responds poorly to escalating analgesia, or is described in catastrophic or all-encompassing language ("nothing works," "the pain is everywhere").

Multidisciplinary management. Physical symptoms are addressed with standard pharmacological and non-pharmacological measures. Psychological distress benefits from counselling, cognitive-behavioural techniques, and, where indicated, pharmacotherapy for anxiety/depression. Medical social workers address financial and family concerns and practical support systems. Spiritual/pastoral care providers help patients explore meaning, reconciliation, and legacy concerns regardless of religious affiliation. Coordinated team meetings and shared documentation ensure a consistent, holistic care plan.

Significance for examinations and practice. The total pain concept underlies the entire philosophy of palliative medicine and the rationale for its multidisciplinary team structure; it is frequently tested and should be explicitly invoked whenever a case describes pain refractory to adequate analgesic escalation.

Conclusion. Effective pain control in advanced illness requires attention to all four domains of total pain, delivered through a coordinated multidisciplinary team rather than physician-led pharmacotherapy alone.

ALGORITHM · Assessment and Management of Total Pain Algo 31 — full page ›



★ KEY POINTS

- Chemical coping (using opioids for emotional distress) signals unaddressed total pain.
- Ask: 'What worries you most?' — often reveals the hidden domain.

MODEL LONG-ESSAY ANSWER · LE 4

Oncologic Emergencies in Palliative Care

Read the text ›

Introduction. Although palliative care emphasises comfort over aggressive intervention, several complications of advanced cancer constitute true emergencies where prompt recognition and treatment can prevent irreversible harm or sudden death, and treatment of these emergencies remains appropriate even in patients receiving palliative-focused care, when consistent with goals of care.

Spinal cord compression. Presents with back pain, limb weakness, sensory level, and sphincter disturbance. Immediate high-dose dexamethasone, urgent whole-spine MRI, and surgical decompression or radiotherapy (depending on operability) are required; outcome depends critically on how quickly weakness is reversed.

Superior vena cava obstruction. Facial/neck swelling, distended neck veins, breathlessness, often from mediastinal lymphadenopathy or lung cancer. Managed with corticosteroids, elevation of the head, and definitive treatment (radiotherapy, chemotherapy, or stenting) depending on histology and urgency.

Hypercalcaemia of malignancy. Confusion, constipation, polyuria; managed with rehydration followed by IV bisphosphonate, guided by goals of care.

Tumour lysis syndrome. Typically after chemotherapy in high-turnover tumours; hyperkalaemia, hyperuricaemia, hyperphosphataemia, hypocalcaemia, and acute kidney injury; managed with hydration, rasburicase/allopurinol, and electrolyte correction.

Massive haemorrhage. From tumour erosion into a major vessel (e.g. head and neck cancers, erosion into carotid); requires pre-emptive discussion with family, dark towels to reduce visual distress, and rapid sedation (e.g. midazolam) as a compassionate response when bleeding is catastrophic and not amenable to intervention.

Neutropenic sepsis and **massive pulmonary embolism** are additional emergencies requiring prompt recognition.

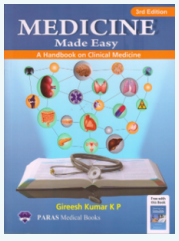
Conclusion. Recognising oncologic emergencies and treating them promptly — within the framework of the patient's overall goals of care — is a core competency for the palliative medicine postgraduate, since timely intervention can prevent otherwise avoidable suffering.

EXAM-ORIENTED THEORY

Oncological emergencies at a glance

Emergency	Key immediate step
Spinal cord compression	Dexamethasone 16 mg + MRI within 24 h
Hypercalcaemia	IV saline + bisphosphonate
SVCO	Steroids, stent, histology
Febrile neutropenia	Antibiotics within 1 hour
Tumour lysis syndrome	Hydration, rasburicase, monitor electrolytes
Catastrophic haemorrhage	Dark towels, midazolam, presence
Raised ICP / seizures	Dexamethasone; benzodiazepine for seizures
Cardiac tamponade	Pericardiocentesis
Airway obstruction	Steroids, stent; palliative sedation if terminal

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MODEL LONG-ESSAY ANSWER · LE 5

Ethical Principles in Palliative Care

Introduction. Palliative care decision-making is grounded in the four classical principles of biomedical ethics — autonomy, beneficence, non-maleficence, and justice — which frequently come into tension at the end of life and require careful, case-by-case balancing.

Autonomy. The right of a competent patient to make informed decisions about their own care, including refusing treatment. This underlies informed consent, truth-telling, advance care planning, and respect for a validly executed Advance Medical Directive.

Beneficence and non-maleficence. The duty to act in the patient's best interest and to avoid causing harm, which in palliative care often means weighing the burden of an intervention (e.g. aggressive chemotherapy, feeding tubes, CPR) against its likely benefit, and withholding or withdrawing treatments judged more burdensome than beneficial.

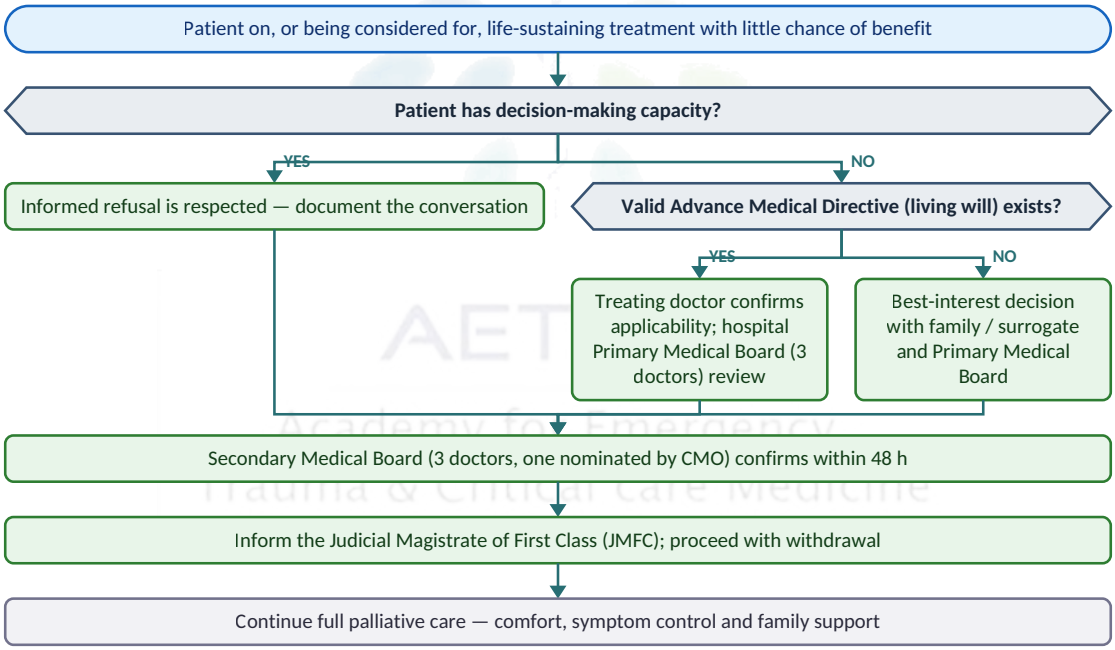
Justice. Fair allocation of limited healthcare resources (opioids, hospice beds, specialist time), particularly salient in resource-constrained settings such as India, where access to palliative care and essential medicines remains inequitable.

The doctrine of double effect. A key applied concept: an action with a good intended effect (symptom relief) that also carries a foreseen but unintended harmful effect (e.g. sedation, theoretical respiratory depression) is ethically permissible provided the intent is relief of suffering, the harmful effect is not the means to the good effect, and the dose used is proportionate to the symptom — this underpins the ethical legitimacy of appropriately titrated opioid and sedative use at the end of life.

Withholding versus withdrawing treatment. Ethically and legally equivalent acts (not euthanasia) when a treatment is judged futile or no longer aligned with the patient's goals, though withdrawing is often psychologically more difficult for families and clinicians.

Conclusion. Ethical palliative care practice requires continuous, explicit application of these principles through shared decision-making with patients and families, well-documented reasoning, and, where needed, input from the institutional ethics committee.

ALGORITHM · Withholding or Withdrawing Life-Sustaining Treatment in India Algo 36 — full page ›



★ KEY POINTS

- Withdrawal of futile treatment is not euthanasia — intent is to stop burdensome treatment, not to cause death.
- The 2023 modification removed the need for magistrate countersignature of living wills (notary/gazetted officer attestation suffices).

MODEL LONG-ESSAY ANSWER · LE 6

Euthanasia, Physician-Assisted Dying, and the Indian Legal Position

Introduction. End-of-life decision-making in India operates within a specific legal framework shaped by the Supreme Court's landmark judgments, which every palliative medicine postgraduate must understand clearly, including the important distinctions between permitted and prohibited practices.

Definitions. *Euthanasia* is the deliberate act of ending a patient's life to relieve suffering, classified as active (a deliberate act causing death, e.g. a lethal injection) or passive (withholding/withdrawing life-sustaining treatment, allowing death to occur naturally). *Physician-assisted suicide* involves a doctor providing the means for a patient to end their own life. *Palliative sedation* and appropriate symptom-relieving opioid use, by contrast, are not euthanasia, since the intent is symptom relief, not death (doctrine of double effect).

The Aruna Shanbaug case (2011). The Supreme Court first permitted passive euthanasia under strict judicial oversight (High Court approval required for each case) for patients in a permanent vegetative state.

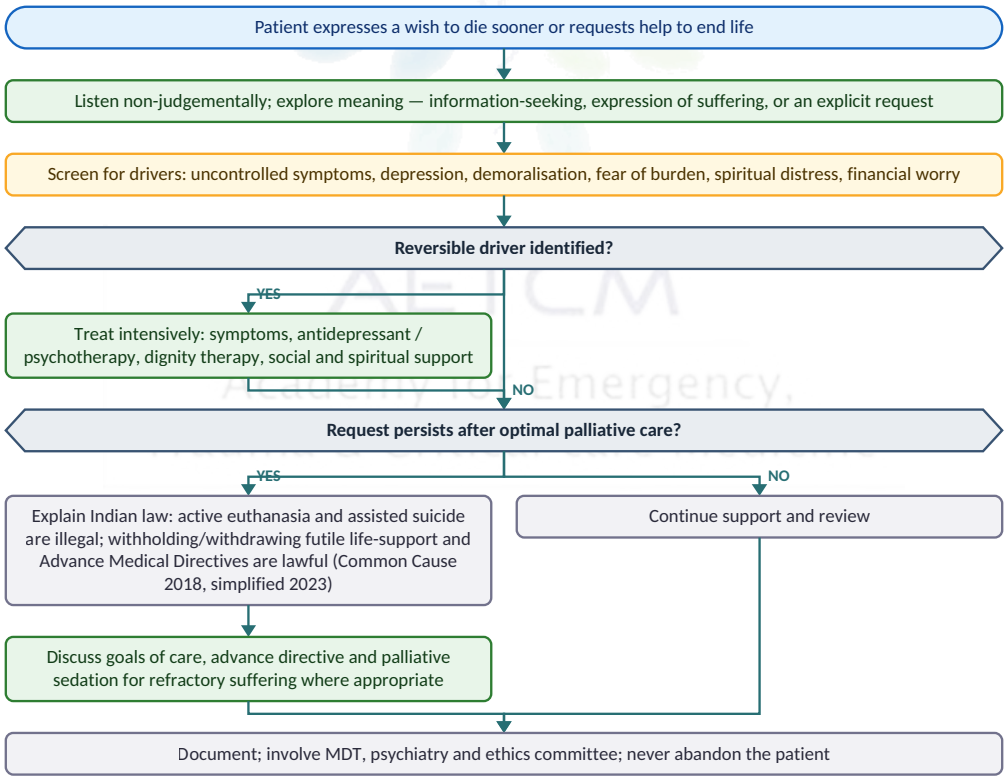
Common Cause vs Union of India (2018). The Supreme Court recognised the right to die with dignity as part of the fundamental right to life under Article 21, legalised passive euthanasia (withholding/withdrawing treatment), and permitted execution of a written Advance Medical Directive (living will) by competent adults specifying their wishes for future treatment if they become incapacitated, subject to a defined certification procedure.

2023 modification. The Supreme Court simplified the cumbersome 2018 procedure for executing and implementing an Advance Medical Directive, reducing the layers of medical board and judicial approval required, making passive euthanasia/withdrawal more practically accessible.

What remains illegal in India. Active euthanasia and physician-assisted suicide remain criminal offences; only passive withdrawal/withholding of futile or disproportionately burdensome treatment, and a valid Advance Medical Directive, are legally sanctioned.

Conclusion. Indian palliative care practice must clearly distinguish lawful passive withdrawal and symptom-focused care (including proportionate sedation) from unlawful active euthanasia, while supporting patients' rights to advance care planning within this evolving legal framework.

ALGORITHM · Approach to a Request for Hastened Death Algo 14 — full page ›



MODEL LONG-ESSAY ANSWER · LE 7

Truth-Telling and Breaking Bad News

Introduction. Communicating a life-limiting diagnosis or deteriorating prognosis is one of the most challenging and frequently examined skills in palliative medicine, requiring both ethical grounding and a structured communication technique.

Why truth-telling matters. Respecting patient autonomy requires that patients be given accurate information to make informed decisions about their remaining life and treatment; withholding information, even when well-intentioned (a common cultural practice in India of informing family but not the patient), can undermine trust, prevent informed consent, and deny patients the opportunity to complete important personal, financial, or spiritual business.

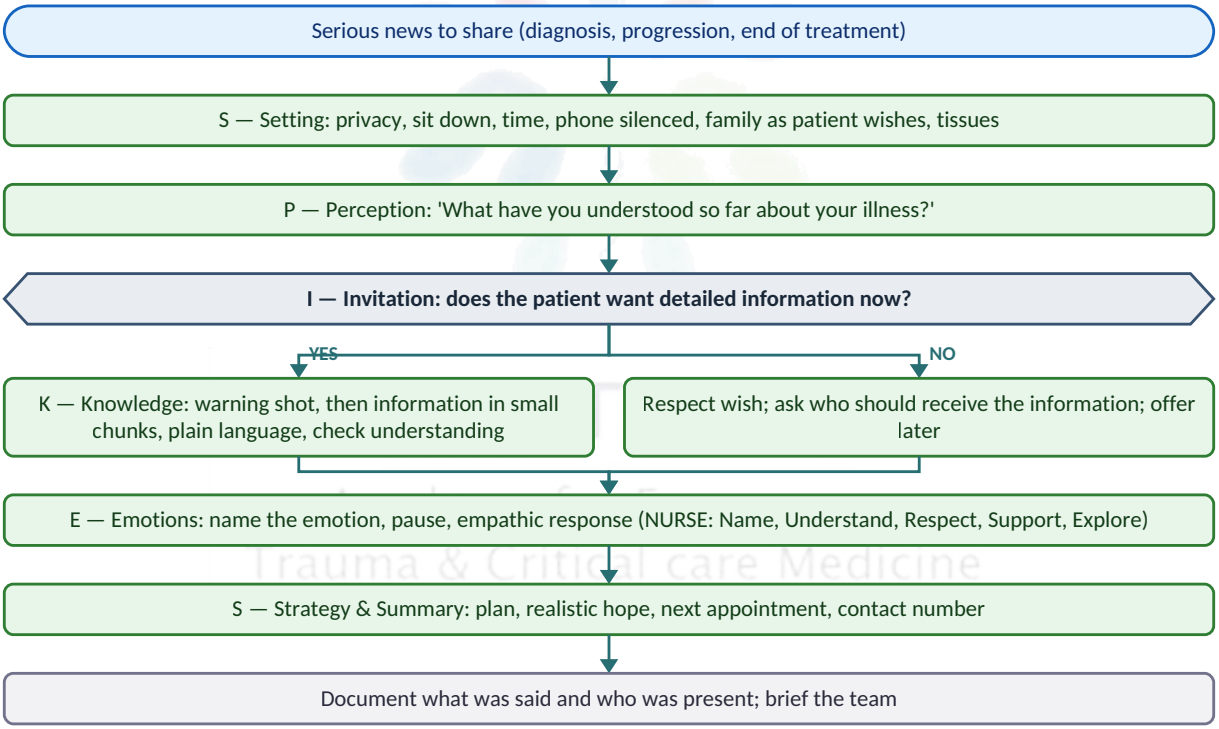
Cultural considerations in the Indian context. Family-centred decision-making is common, and clinicians must sensitively navigate between respecting family wishes and the patient's own right to information, typically by exploring with the patient first how much they wish to know and whether they want family involved in discussions, rather than assuming either extreme.

The SPIKES protocol provides a structured, six-step framework: Setting up the interview (privacy, time, seating); Perception (assessing what the patient already understands); Invitation (gauging how much information is wanted); Knowledge (delivering information in small chunks, in plain language, with a warning shot); Emotions (responding empathetically to the patient's reaction, using techniques like naming, understanding, respecting, exploring); and Strategy/Summary (agreeing a follow-up plan).

Common pitfalls. Delivering too much information at once, using euphemisms that obscure meaning, failing to check understanding, and moving to logistics before addressing emotion.

Conclusion. Skilful, honest, and empathetic communication of bad news, delivered using a structured approach such as SPIKES and adapted to the patient's cultural context and expressed wishes, is a core, assessable clinical competency in palliative medicine.

ALGORITHM · Breaking Bad News — the SPIKES Protocol Algo 12 — full page ›



★ KEY POINTS

- Silence is a skill — allow it after the news.
- In India, families often request non-disclosure — explore their fears and the patient's wishes, don't simply comply.

MODEL LONG-ESSAY ANSWER · LE 8

Palliative Sedation: Indications, Ethics and Practice

Introduction. Palliative sedation is the monitored use of sedative medication to relieve otherwise intractable suffering in patients approaching the end of life, reserved for refractory symptoms that have not responded to all other appropriate interventions.

Indications. Refractory physical symptoms — severe agitated terminal delirium, intractable pain, dyspnoea, or seizures — that cannot be adequately controlled despite exhaustive appropriate treatment, in a patient with an irreversible, advanced, terminal condition. Existential/psychological suffering as a sole indication remains more controversial and requires particularly careful multidisciplinary assessment.

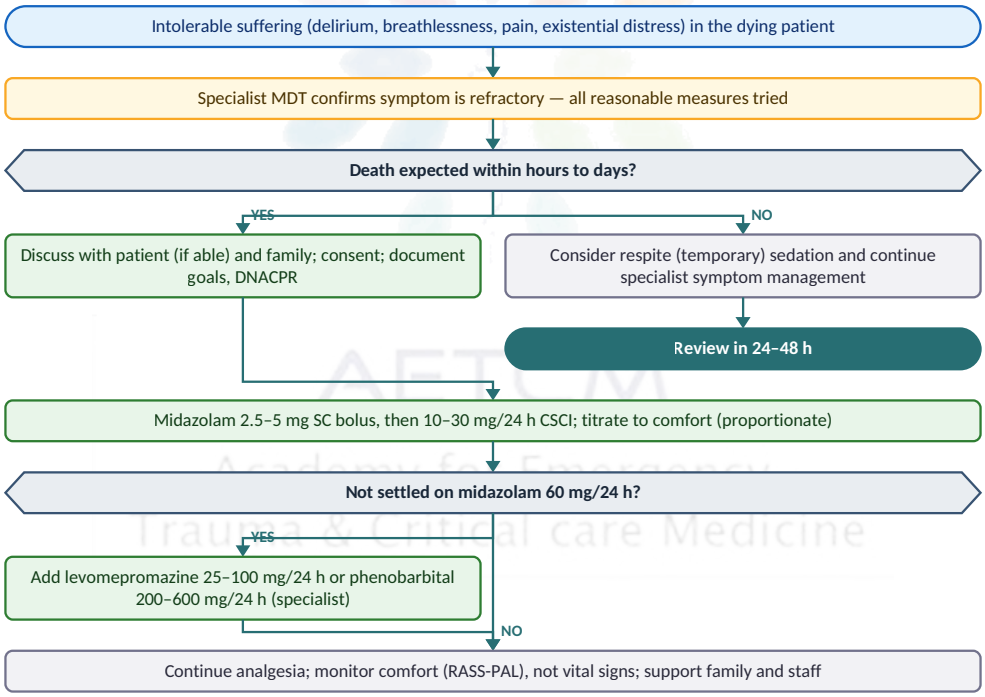
Principles of practice. Sedation should be proportionate — starting with the lowest dose likely to relieve distress and titrating upward only as needed (proportionate sedation), rather than immediately inducing deep unconsciousness, except in a true crisis (e.g. catastrophic haemorrhage) where rapid sedation is compassionate. Informed consent (or, where incapacity exists, appropriate surrogate/family discussion) should be obtained wherever feasible, and the decision-making process clearly documented.

Ethical distinction from euthanasia. Palliative sedation is justified under the doctrine of double effect — the intent is relief of refractory symptoms, not death; sedative doses are titrated to symptom control, not to hasten death; and, critically, evidence shows appropriately titrated palliative sedation does not shorten survival compared with standard symptom management.

Practical considerations. Commonly used agents include midazolam (first-line in most protocols), levomepromazine, and phenobarbital for refractory cases; nutrition and hydration decisions are made separately and individually, generally withheld once sedation is established for comfort, as artificial nutrition/hydration in the actively dying confers no benefit and may increase secretions/discomfort.

Conclusion. Palliative sedation is an ethically sound, evidence-based intervention of last resort for refractory end-of-life suffering, clearly distinguishable from euthanasia by its intent, proportionality, and effect on survival.

ALGORITHM · Palliative Sedation Algo 37 — full page ›



★ KEY POINTS

- Proportionate sedation does not hasten death when appropriately titrated (Maltoni 2012).
- Doctrine of double effect: intent is relief of suffering.

MODEL LONG-ESSAY ANSWER · LE 9

Organization and Models of Palliative Care Delivery in India

Introduction. India faces an enormous and growing need for palliative care, driven by a rising cancer burden, an ageing population, and increasing chronic non-communicable disease, against which access remains highly inequitable and services are unevenly distributed.

Levels of care. *Primary level* — basic symptom management and referral by general physicians and primary health centres, ideally integrated into routine care. *Secondary/specialist level* — dedicated palliative care units/ departments in district and tertiary hospitals providing specialist consultation, inpatient and outpatient services. *Tertiary/academic level* — teaching hospitals with MD/DNB training programmes, research, and complex symptom management.

Settings of care. Inpatient hospice and hospital-based units for complex symptom control; home-based palliative care, which delivers the majority of care in India given limited bed capacity and patient preference to remain at home; day-care centres; and outpatient clinics.

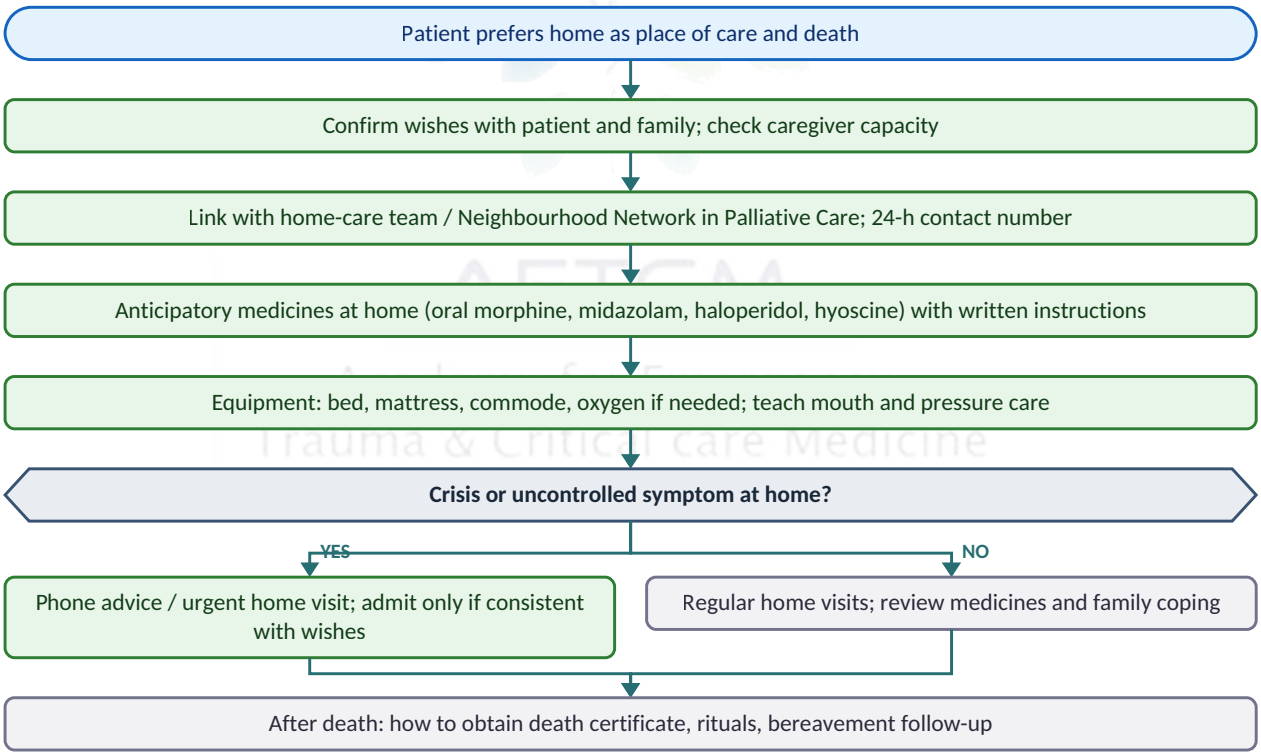
The Kerala/community-based model. Kerala pioneered a community-owned, volunteer-driven, neighbourhood network model of palliative care (Neighbourhood Network in Palliative Care) in which trained local volunteers identify and support patients at home, supported by professional teams for clinical needs — widely cited internationally as a scalable, low-cost model achieving some of the highest palliative care coverage in the country.

National policy framework. The National Programme for Palliative Care (NPPC), incorporation of palliative care into the National Health Mission, and amendments to the Narcotic Drugs and Psychotropic Substances (NDPS) Act (2014) simplifying the licensing process for opioid access by recognised healthcare institutions, have improved (though not yet resolved) the historic problem of "opiophobia" and poor morphine availability in India.

Challenges. Workforce shortage, inconsistent opioid availability across states, low public awareness, uneven integration with oncology and primary care, and wide rural-urban disparity in service availability.

Conclusion. Scaling palliative care in India requires a layered system combining specialist tertiary services with scalable, low-cost community and home-based models such as Kerala's, supported by continued policy reform in opioid access and workforce training.

ALGORITHM · Planning for Death at Home Algo 61 — full page ›



MODEL LONG-ESSAY ANSWER · LE 10

Grief and Bereavement: Theories and Management

Introduction. Supporting the bereaved is an integral part of palliative care, extending beyond the patient's death to structured follow-up with the family, grounded in an understanding of normal versus complicated grief.

Theoretical models. Kubler-Ross's five stages (denial, anger, bargaining, depression, acceptance), though originally describing the dying patient's own adjustment rather than bereavement, are widely (if imperfectly) applied to grief; importantly these stages are not strictly linear or universal. Worden's four tasks of mourning — accepting the reality of the loss, processing the pain of grief, adjusting to a world without the deceased, and finding an enduring connection while moving forward with life — offer a more actionable framework for clinical support. The dual-process model describes healthy grieving as oscillating between loss-oriented coping (grief work) and restoration-oriented coping (attending to practical life changes), rather than requiring sequential completion of "stages."

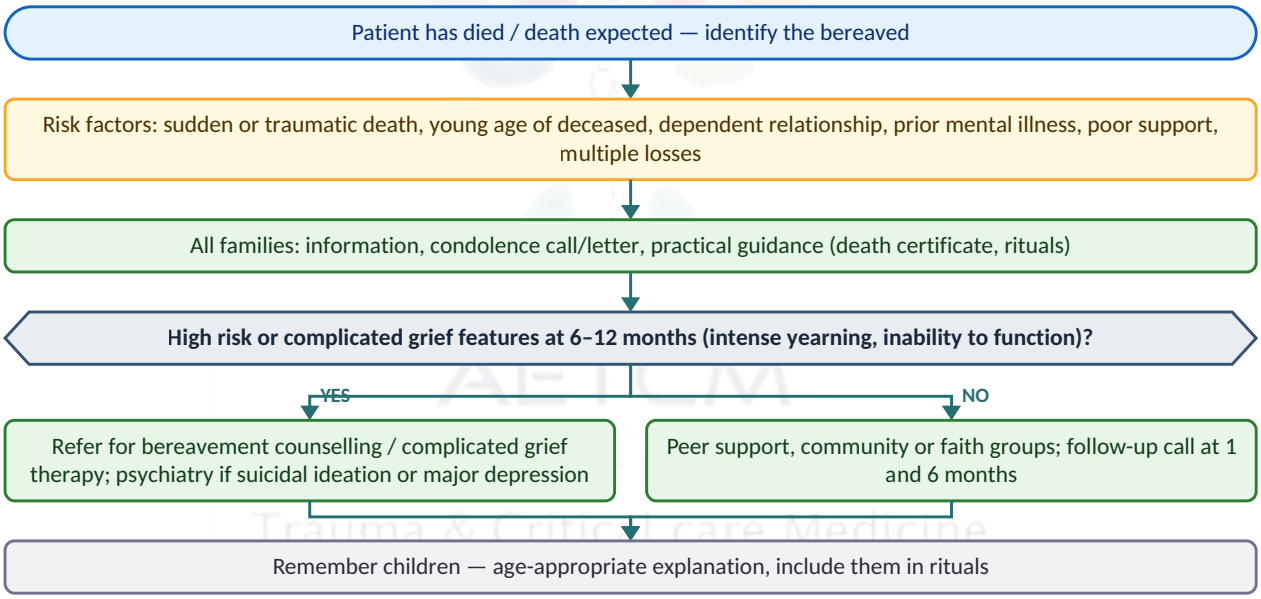
Normal versus complicated grief. Normal grief, though intensely painful, gradually integrates over time without major functional impairment. Complicated/prolonged grief disorder is characterised by persistent, intense yearning and functional impairment extending well beyond expected cultural and social norms (generally >6–12 months), warranting specialist mental health referral.

Risk factors for complicated grief. Sudden or traumatic death, ambivalent or highly dependent relationships, lack of social support, pre-existing mental illness, multiple losses, and a death perceived as poorly managed or with unresolved conflict.

Bereavement support in practice. Pre-death preparation (anticipatory guidance), presence and practical support around the time of death, follow-up contact (calls, condolence visits, or structured bereavement programmes) at intervals after death, and referral to counselling or psychiatric services when complicated grief is identified.

Conclusion. Effective bereavement care requires understanding normal grief trajectories, proactively identifying those at risk of complicated grief, and offering structured, culturally sensitive follow-up as a core extension of the palliative care team's responsibility to the family.

ALGORITHM · Bereavement Risk Assessment and Support Algo 38 — full page ›



★ KEY POINTS

- Prolonged grief disorder (ICD-11): persistent grief > 6 months with functional impairment.
- Kübler-Ross stages are not linear — dual process model is more useful.

FIGURE Five stages of grief (Kübler-Ross) — active/outward vs passive/inward, not linear (Section 7.22) p.487 ›

MODEL LONG-ESSAY ANSWER · LE 11

Spiritual Care in Palliative Medicine

Read the text ›

Introduction. Spiritual care addresses the existential dimension of suffering — questions of meaning, purpose, hope, forgiveness, and connection — recognised as the fourth domain of Cicely Saunders' total pain concept and a core, assessable component of holistic palliative care, independent of specific religious affiliation.

Spirituality versus religion. Spirituality is a broader, universal human search for meaning and connection (to self, others, nature, or the transcendent), which may or may not be expressed through organised religious belief or practice; palliative care spiritual assessment addresses both religious and secular/existential dimensions.

Common sources of spiritual distress. Fear of the unknown/death, feelings of guilt or unfinished business, loss of meaning or purpose, anger at a higher power or at the injustice of illness, and disruption of previously sustaining religious practice or community.

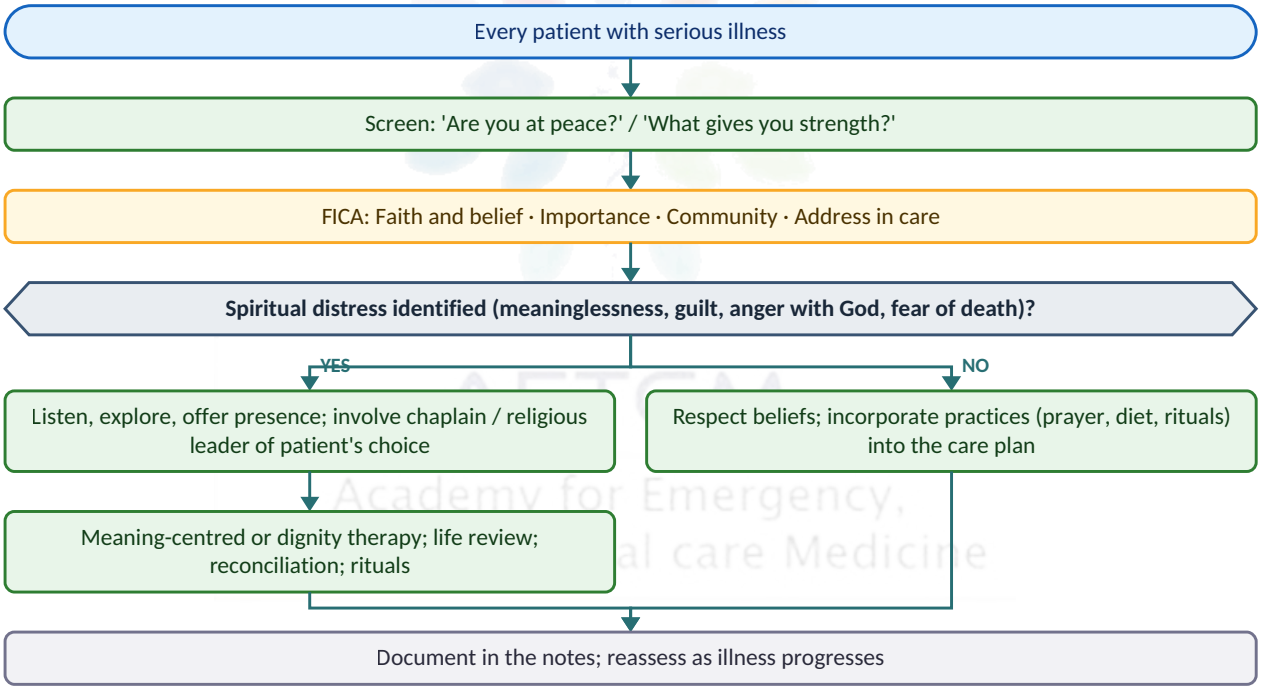
Assessment tools. Structured spiritual history tools such as FICA (Faith, Importance, Community, Address in care) and HOPE (sources of Hope, Organised religion, Personal spirituality, Effects on care) provide a practical framework for eliciting spiritual needs in routine consultations without requiring specialist training.

Role of the chaplain/spiritual care provider. Specialist spiritual care providers offer deeper exploration, religious rituals as desired, legacy work, and facilitation of reconciliation, complementing rather than replacing the spiritual sensitivity expected of all team members.

Practical integration. Simple interventions — unhurried presence, active listening, facilitating visits from religious leaders or family reconciliation, and respecting end-of-life rituals important to the patient's faith or culture — are within every clinician's remit and often provide profound comfort.

Conclusion. Spiritual care, delivered through routine assessment and specialist referral where needed, is an essential, evidence-valued component of holistic palliative care that directly addresses one of the four domains of total suffering.

ALGORITHM · Spiritual Assessment (FICA) and Care Algo 39 — full page ›



★ KEY POINTS

- Spirituality is broader than religion — it is about meaning and connection.
- HOPE and SPIRIT are alternative assessment tools.

MODEL LONG-ESSAY ANSWER · LE 12

Principles of Pediatric Palliative Care

Introduction. Pediatric palliative care is a distinct discipline addressing the needs of children with life-limiting or life-threatening conditions and their families, differing substantially from adult palliative care in disease trajectory, communication, pharmacology, and family dynamics.

Disease trajectories. Unlike the relatively predictable decline seen in many adult cancers, paediatric life-limiting conditions span diverse trajectories: progressive malignancies, life-limiting genetic/metabolic/ neurodegenerative conditions (often with very long, uncertain courses), severe neurological impairment from birth, and conditions where cure is possible but the risk of treatment failure is high — care may therefore run in parallel with curative treatment for months to years.

Communication with children and families. Information must be tailored to the child's developmental stage, using age-appropriate language and, where possible, involving the child directly in decisions about their own care; parents are central partners and decision-makers, and the whole family unit (including siblings) is the unit of care.

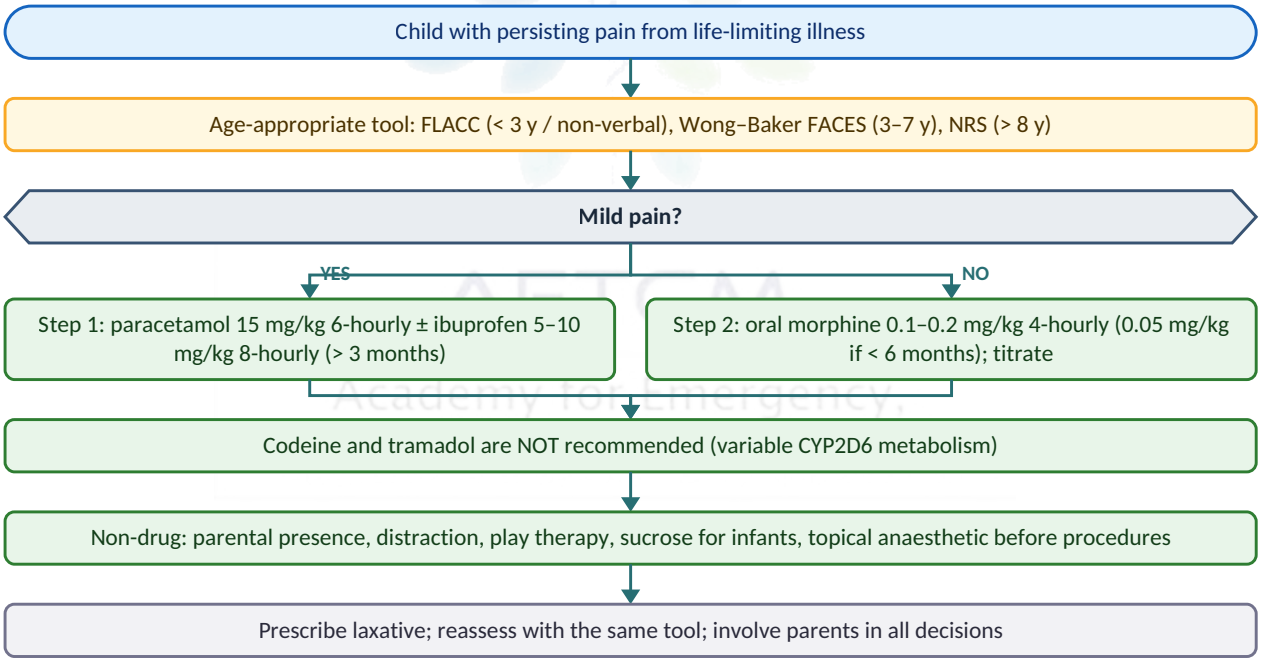
Pain and symptom assessment. Requires age-appropriate validated tools — observational scales (e.g. FLACC, CRIES) for infants/non-verbal children, self-report scales (Wong-Baker FACES, numerical rating) for older children — and particular attention to under-recognition of pain in children, historically a major gap in care.

Pharmacological considerations. Weight-based dosing, age-appropriate formulations (liquid preparations, avoiding transdermal patches in very young/cachectic children), and caution regarding drugs with limited paediatric safety data; the WHO's two-step approach for children uses non-opioids for mild pain and moves directly to strong opioids for moderate-severe pain.

Family and sibling support. Siblings are often an under-recognised group needing age-appropriate explanation, involvement, and bereavement support; parental psychological burden is profound and requires proactive attention, including respite care where available.

Conclusion. Pediatric palliative care demands developmentally-sensitive communication, specialised symptom assessment and pharmacology, and whole-family support across often long and uncertain illness trajectories distinct from the adult model.

ALGORITHM · Pain Management in Children Algo 32 — full page ›



MODEL LONG-ESSAY ANSWER · LE 13

Palliative Care in Non-Malignant Disease

Introduction. Although palliative medicine developed historically around cancer care, the majority of the global need for palliative care arises from non-malignant, life-limiting conditions — cardiovascular disease, chronic respiratory disease, dementia, and end-stage organ failure — which remain underserved relative to cancer patients.

Disease-trajectory differences from cancer. Cancer typically shows a relatively predictable period of stable function followed by a rapid terminal decline, making prognostication and timing of palliative referral comparatively easier. Organ failure (heart failure, COPD, ESRD) follows a trajectory of gradual decline punctuated by acute exacerbations/ hospitalisations, with death often occurring unexpectedly during an exacerbation, making prognostication difficult and referral frequently too late. Dementia and frailty show a prolonged, slow decline over years, with late and gradual loss of capacity.

Heart failure. Breathlessness, fatigue, and fluid overload require integration of palliative symptom control with ongoing disease-directed therapy (diuretics, guideline-directed medical therapy); discussions about deactivating implantable defibrillators become important near the end of life to avoid distressing shocks during dying.

COPD. Refractory breathlessness managed with low-dose opioids and non-pharmacological measures, alongside continued inhaled therapy; advance care planning regarding future ventilatory support is important given risk of acute-on-chronic respiratory failure.

End-stage renal disease. Conservative (non-dialysis) management pathways, symptom control (pruritus, fatigue, pain with caution for renally-cleared drugs), and advance care planning for patients choosing not to pursue or to withdraw from dialysis.

Dementia. Careful hand feeding over tube feeding, proactive advance care planning before capacity is lost, and family support for a prolonged, often ambiguous dying process.

Barriers to non-malignant palliative care. Prognostic uncertainty, under-recognition of the life-limiting nature of these conditions by treating specialists, and historical association of palliative care exclusively with cancer and imminent death.

Conclusion. Expanding palliative care beyond cancer requires trajectory-specific symptom management strategies and earlier integration alongside disease-directed treatment, supported by proactive, uncertainty-tolerant advance care planning.

EXAM-ORIENTED THEORY

Triggers for palliative care in non-malignant disease

Condition	Indicators (SPICT / GSF)
Heart failure	NYHA III–IV, ≥ 2 admissions/yr, breathlessness at rest despite optimal therapy
COPD	FEV1 < 30%, home oxygen, ≥ 3 admissions/yr, breathless at rest
CKD	eGFR < 15 not for dialysis, or stopping dialysis
Liver disease	Child–Pugh C, refractory ascites, encephalopathy, not transplant candidate
Dementia	Unable to walk/talk/feed, recurrent infections, weight loss (FAST 7)
Neurological (MND, Parkinson's)	Dysphagia, aspiration, respiratory failure, progressive decline
Frailty	Bed-bound, weight loss, multiple falls

MODEL LONG-ESSAY ANSWER · LE 14

The Multidisciplinary Team in Palliative Care

Read the text ›

Introduction. Because suffering in advanced illness spans physical, psychological, social, and spiritual domains (total pain), effective palliative care is inherently a team endeavour, requiring coordinated input from multiple disciplines rather than physician-led care alone.

Core team members and roles. The *physician* leads diagnosis, symptom pharmacotherapy, and complex decision-making. The *nurse* provides continuous assessment, symptom monitoring, wound and stoma care, medication administration, and is often the primary point of contact and trust for patients and families. The *medical social worker* addresses financial assistance, family dynamics, discharge planning, and practical/legal issues (e.g. guardianship, benefits). The *counsellor/psychologist* manages psychological distress, anxiety, depression, and coping. The *chaplain/spiritual care provider* addresses existential and religious needs. *Physiotherapists and occupational therapists* support mobility, function, and adaptive equipment needs. *Pharmacists* assist with complex polypharmacy, opioid conversion calculations, and drug interaction review. *Trained volunteers*, particularly prominent in community-based Indian models, extend reach into the home and community.

Why a team approach is essential. No single discipline has the full skill set to address total pain; regular team meetings allow shared formulation of a holistic care plan, reduce the risk of fragmented or conflicting advice to the family, and distribute the emotional burden of care among providers, reducing burnout.

Coordination mechanisms. Shared documentation, regular case conferences, clearly defined referral pathways, and a designated key worker/care coordinator for each patient/family to ensure continuity.

Challenges in resource-limited settings. Many Indian centres operate with a truncated team (often just a physician and nurse), making volunteer and community partnerships (as in the Kerala model) especially valuable to approximate full multidisciplinary coverage.

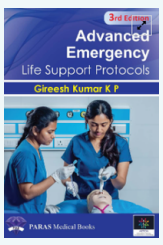
Conclusion. The multidisciplinary team structure is not an optional enhancement but a structural necessity flowing directly from the total pain concept, and its effective coordination is itself a core competency for the palliative medicine postgraduate.

EXAM-ORIENTED THEORY

Roles in the palliative MDT

Member	Contribution
Palliative physician	Diagnosis, complex symptom control, prognosis, decisions
Palliative nurse	Assessment, continuity, home care, family education
Medical social worker	Finances, schemes, family issues, discharge planning
Psychologist / counsellor	Anxiety, depression, adjustment, bereavement
Physiotherapist / OT	Mobility, fatigue, lymphoedema, aids and adaptations
Pharmacist	Opioid access, interactions, formulations
Chaplain / spiritual care	Meaning, rituals, spiritual distress
Volunteers	Community link, companionship (Kerala NNPC model)

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MODEL LONG-ESSAY ANSWER · LE 15

Opioid Rotation and Equianalgesic Dosing: Principles and Practice

Read the text ›

Introduction. Opioid rotation — switching from one opioid (or route) to another — is a core clinical skill used when analgesia is inadequate despite appropriate titration, or when adverse effects (sedation, neurotoxicity, myoclonus, nausea) limit further dose increase, exploiting incomplete cross-tolerance between different opioids and their differing side-effect profiles.

Indications for rotation. Poor analgesia despite adequate dose titration of the current opioid; intolerable adverse effects; need to change route (e.g. oral to transdermal for a patient who can no longer swallow); organ dysfunction (renal/hepatic) making the current opioid less safe; and cost/availability considerations.

Principle of equianalgesic dosing. Standardised conversion tables (referenced to oral morphine as the benchmark) estimate the dose of a new opioid expected to provide comparable analgesia to the current opioid dose; these ratios are derived mainly from single-dose studies and carry significant inter-individual variability, so must be applied as a starting estimate, not an exact prescription.

Incomplete cross-tolerance. Because tolerance developed to one opioid does not fully transfer to a different opioid (acting with a somewhat different receptor-binding profile), the calculated equianalgesic dose of the new opioid is conventionally reduced by 25–50% to avoid overdose, with a smaller reduction if pain remains severe and a larger reduction if the patient is frail, elderly, or has significant comorbidity.

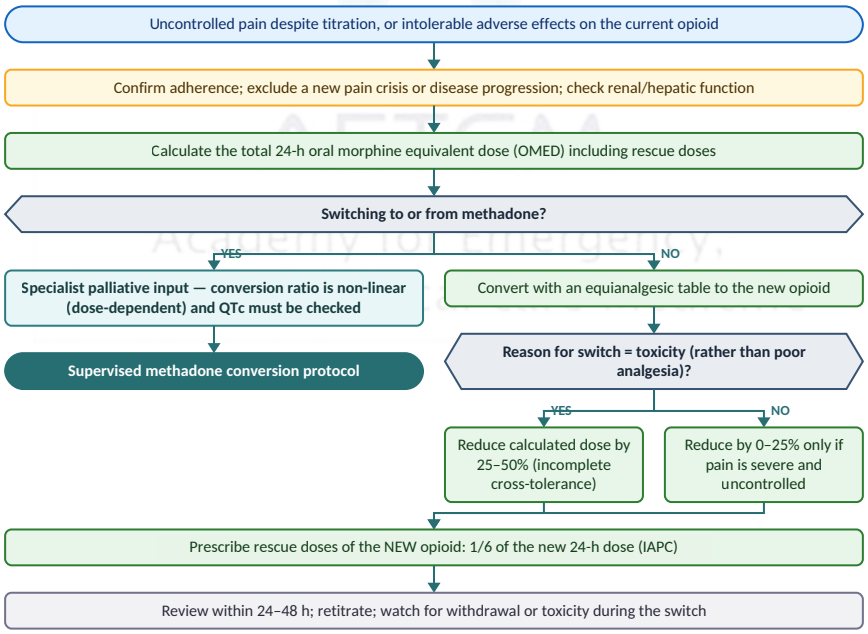
Special case — methadone. Methadone conversion is markedly non-linear — its relative potency compared with morphine increases substantially at higher pre-rotation morphine doses — and it has a long, variable half-life with risk of delayed accumulation and QT prolongation; methadone rotation should only be performed by or with specialist/consultant guidance, typically with a more conservative stop-and-start or gradual overlap protocol rather than a simple one-step conversion.

Transdermal fentanyl/buprenorphine conversions require the patient to already be opioid-tolerant and stable on oral opioids before initiation, given the slow onset and offset of the patch.

Practical safeguards. Always provide an appropriate breakthrough/rescue dose of the new opioid, review within 24–48 hours, and titrate further based on response, rather than assuming the calculated dose is final.

Conclusion. Safe opioid rotation requires accurate equianalgesic calculation, deliberate dose reduction for incomplete cross-tolerance, special caution with methadone, and close early follow-up — making it one of the most practically important and frequently examined skills in palliative pharmacotherapy.

ALGORITHM · Opioid Rotation Algo 2 — full page ›



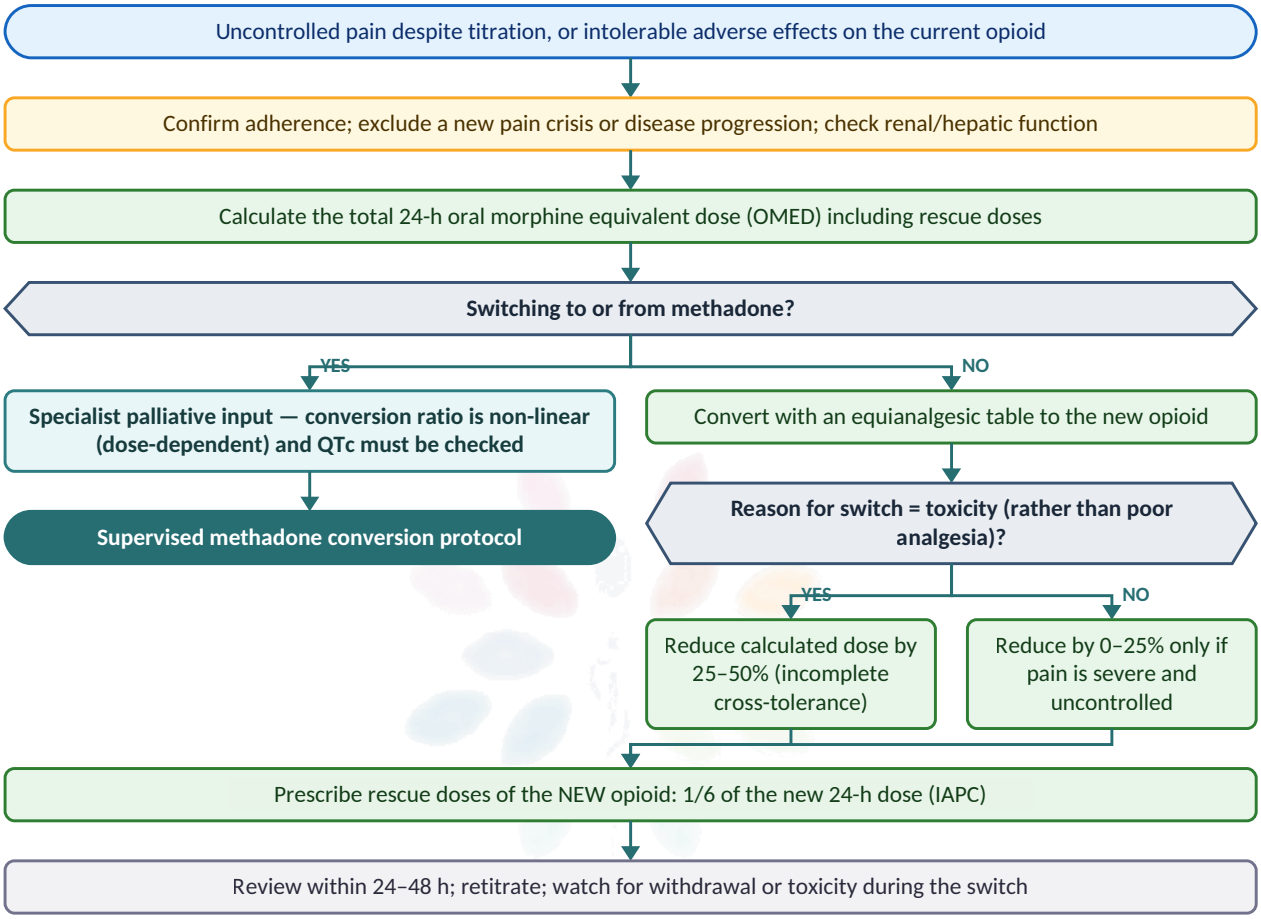
MODEL SHORT-ESSAY ANSWER · SE 1

Opioid Rotation

Read the text ›

Opioid rotation is switching from one opioid (or route) to another because of inadequate analgesia despite titration, or intolerable adverse effects, exploiting incomplete cross-tolerance between opioids. The new dose is calculated using an equianalgesic conversion table referenced to oral morphine, then reduced by 25–50% for incomplete cross-tolerance (less reduction if pain remains severe). A breakthrough dose of the new opioid is provided and the patient reviewed within 24–48 h. Methadone rotation is an exception — its conversion ratio is non-linear and rises with pre-rotation dose, so it should only be performed under specialist guidance.

ALGORITHM · Opioid Rotation Algo 2 — full page ›



★ KEY POINTS

- ▸ Fentanyl patch: start new patch and stop the old opioid at the appropriate time (12 h overlap for SR morphine).
- ▸ Renal failure is the commonest reason to rotate from morphine — choose fentanyl, buprenorphine or alfentanil.

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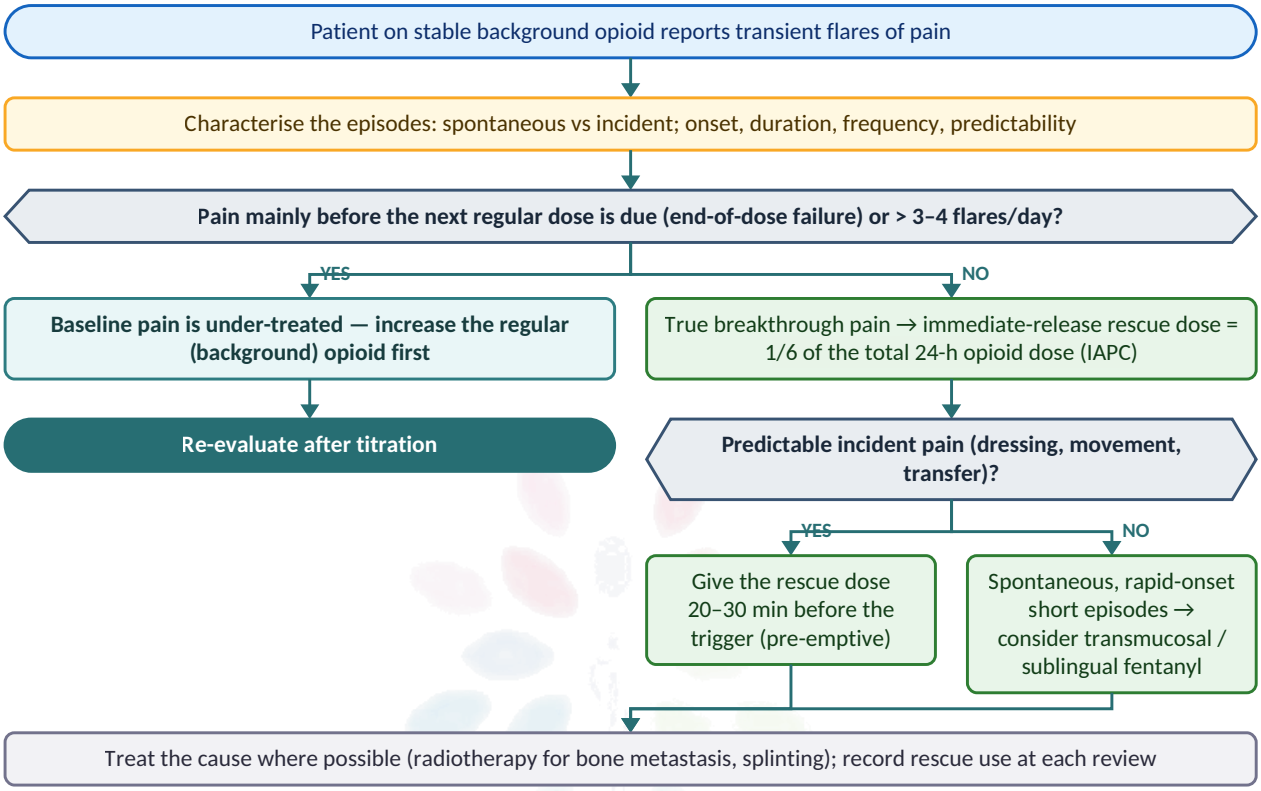
MODEL SHORT-ESSAY ANSWER · SE 2

Breakthrough Pain

Read the text ›

Breakthrough pain is a transient exacerbation of pain occurring despite adequate background (baseline) analgesia. It may be spontaneous (unpredictable) or incident (triggered by movement/activity/procedures), and must be distinguished from end-of-dose failure (inadequate baseline control), which requires increasing the background dose rather than adding rescue doses. True breakthrough pain is managed with an immediate-release opioid rescue dose, typically 1/6 of the total 24-h opioid dose (IAPC), given as needed, or pre-emptively 20– 30 minutes before a predictable trigger. Rapid-onset transmucosal/sublingual fentanyl preparations suit very rapid, short-lived episodes.

ALGORITHM · Approach to Breakthrough Pain Algo 3 — full page ›



★ KEY POINTS

- Typical breakthrough episode peaks in 3–5 min and lasts < 30 min — oral morphine (onset 20–30 min) is often too slow.
- Transmucosal fentanyl doses are titrated independently, not as a % of background dose.

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MODEL SHORT-ESSAY ANSWER · SE 3

Equianalgesic Conversion

Read the text ›

The conversions below follow the **IAPC method** (IAPC Textbook, 5th ed., ch. 6). Every calculation starts from the **total 24-hour oral morphine dose (M_{24})**, including rescue doses taken. Equianalgesic ratios are approximate starting points, so review the patient within 24–48 h. The full working, with the equation before each calculation, is in 7.17 · p.479.

EXAM-ORIENTED THEORY

Equianalgesic rules (IAPC)

Conversion	Equation	Example
Codeine → oral morphine	Morphine/24 h = codeine/24 h ÷ 10	40 mg 4-hourly = 240 mg → 24 mg/24 h
IR 4-hourly → SR 12-hourly	SR dose = M_{24} ÷ 2, twice daily	20 mg 4-hourly (120 mg) → SR 60 mg bd
Breakthrough (rescue) dose	M_{24} ÷ 6 (= the 4-hourly dose)	SR 60 mg bd (120 mg) → IR 20 mg PRN
Oral → SC morphine (CSCI)	SC_{24} = M_{24} ÷ 2 (SC is 2× as potent)	120 mg oral → 60 mg SC/24 h
SC 24-h → intermittent SC	SC dose 4-hourly = SC_{24} ÷ 6	60 mg/24 h → 10 mg SC 4-hourly
Oral morphine → fentanyl patch	M_{24} ÷ 100 = mg/24 h; × 1000 ÷ 24 = µg/h (≈ M_{24} ÷ 2.4)	180 mg → 1.8 mg → 1800 µg ÷ 24 = 75 µg/h
Rule of thumb (IAPC)	M_{24} ÷ 3 → nearest patch strength	180 ÷ 3 = 60 → 50 or 75 µg/h
Fentanyl patch → oral morphine	M_{24} = patch (µg/h) × 2.4	25 µg/h → 60 mg/24 h; rescue 10 mg
Oral tramadol → oral morphine	÷ 10 (book standard; IAPC gives no ratio)	400 mg tramadol ≈ 40 mg morphine

Not covered by IAPC (ratios unchanged from this book)	Rule
Oral morphine → oral oxycodone	Divide by 1.5–2
Oral oxycodone → SC oxycodone	Divide by 2
Oral morphine → oral hydromorphone	Divide by 5
SC morphine → SC alfentanil	Divide by 10

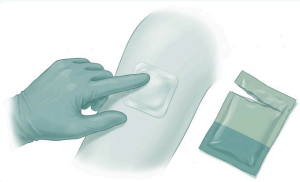
EXAM-ORIENTED THEORY

Worked example (IAPC method)

Step	Calculation
Patient on SR morphine 60 mg bd + 3 × 10 mg rescue	M_{24} = 60 × 2 + 3 × 10 = 150 mg
New breakthrough dose	150 ÷ 6 = 25 mg IR morphine as needed
Unable to swallow → CSCI	SC_{24} = 150 ÷ 2 = 75 mg morphine/24 h; PRN SC = 75 ÷ 6 ≈ 12.5 mg
Or → fentanyl patch	150 × 10 ÷ 24 = 62.5 µg/h; rule of thumb 150 ÷ 3 = 50 → 50 µg/h patch (+ IR rescue 25 mg)
If on IR morphine instead (e.g. 20 mg 4-hourly)	M_{24} = 20 × 6 = 120 mg → rescue 20 mg · SC 60 mg/24 h · patch 120 × 10 ÷ 24 = 50 µg/h (keep IR 4-hourly for the first 12 h)
Switching because of toxicity	Start lower (reduce by 25–50%) and retitrate — see opioid rotation, p.270

★ CLINICAL PEARLS & EXAM FOCUS

- Apply the fentanyl patch with the last SR dose, or continue 4-hourly IR morphine for the first 12 h — levels peak at 12–24 h. The same M_{24} rules apply whether the patient is on IR or SR morphine.
- Syringe-driver algorithm (Algo 28): p.296; full IAPC working with equations: p.479.



ILLUSTRATION

Applying a fentanyl patch

- Clean, dry, hairless, intact skin (upper arm or chest); press firmly for 30 seconds.
- Change every 72 hours, rotating sites; avoid heat pads and treat fever (absorption rises).
- Effect takes 12–24 h to build — continue the previous opioid during that time.

MODEL SHORT-ESSAY ANSWER · SE 4

Adjuvant Analgesics

Read the text ›

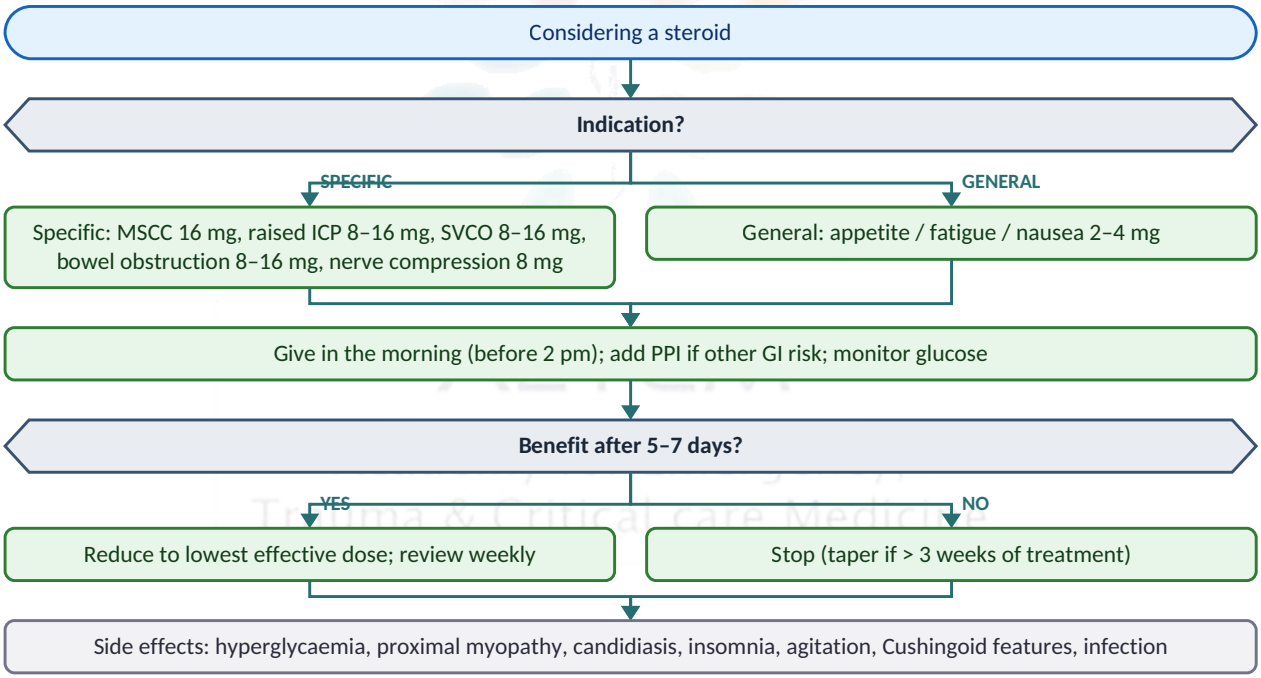
Adjuvant analgesics are drugs whose primary indication is not pain but which have analgesic effects in specific pain types, usable at any step of the WHO ladder. Anticonvulsants (gabapentin, pregabalin) and antidepressants (amitriptyline, duloxetine) are first-line for neuropathic pain. Corticosteroids (dexamethasone) relieve pain from nerve/cord compression, raised intracranial pressure, and inflammatory bone pain. Bisphosphonates/denosumab reduce bone pain and skeletal events. Ketamine (an NMDA-receptor antagonist) is used for refractory neuropathic pain. Muscle relaxants help pain with a significant spasm component. Choosing the right adjuvant for the pain mechanism, rather than escalating opioids alone, is central to rational multimodal analgesia.

EXAM-ORIENTED THEORY

Adjuvant analgesics by pain type

Pain type	Adjuvant	Starting dose
Neuropathic	Amitriptyline / nortriptyline	10 mg nocte
Neuropathic	Gabapentin / pregabalin	300 mg nocte / 75 mg bd
Neuropathic	Duloxetine	30 mg daily
Nerve / cord compression, liver capsule, raised ICP	Dexamethasone	8–16 mg/day
Bone	NSAID; bisphosphonate / denosumab	Ibuprofen 400 mg tds; zoledronate 4 mg
Colic	Hyoscine butylbromide	20 mg SC PRN; 60–120 mg/24 h
Muscle spasm	Baclofen / diazepam	5 mg tds / 2–5 mg
Refractory / hyperalgesia	Ketamine (specialist)	10–25 mg oral tds

ALGORITHM · Use of Corticosteroids in Palliative Care Algo 60 — full page ›



★ KEY POINTS

- Proximal myopathy appears after 2–4 weeks and limits mobility.
- Steroid-induced hyperglycaemia peaks in the afternoon — check evening glucose.

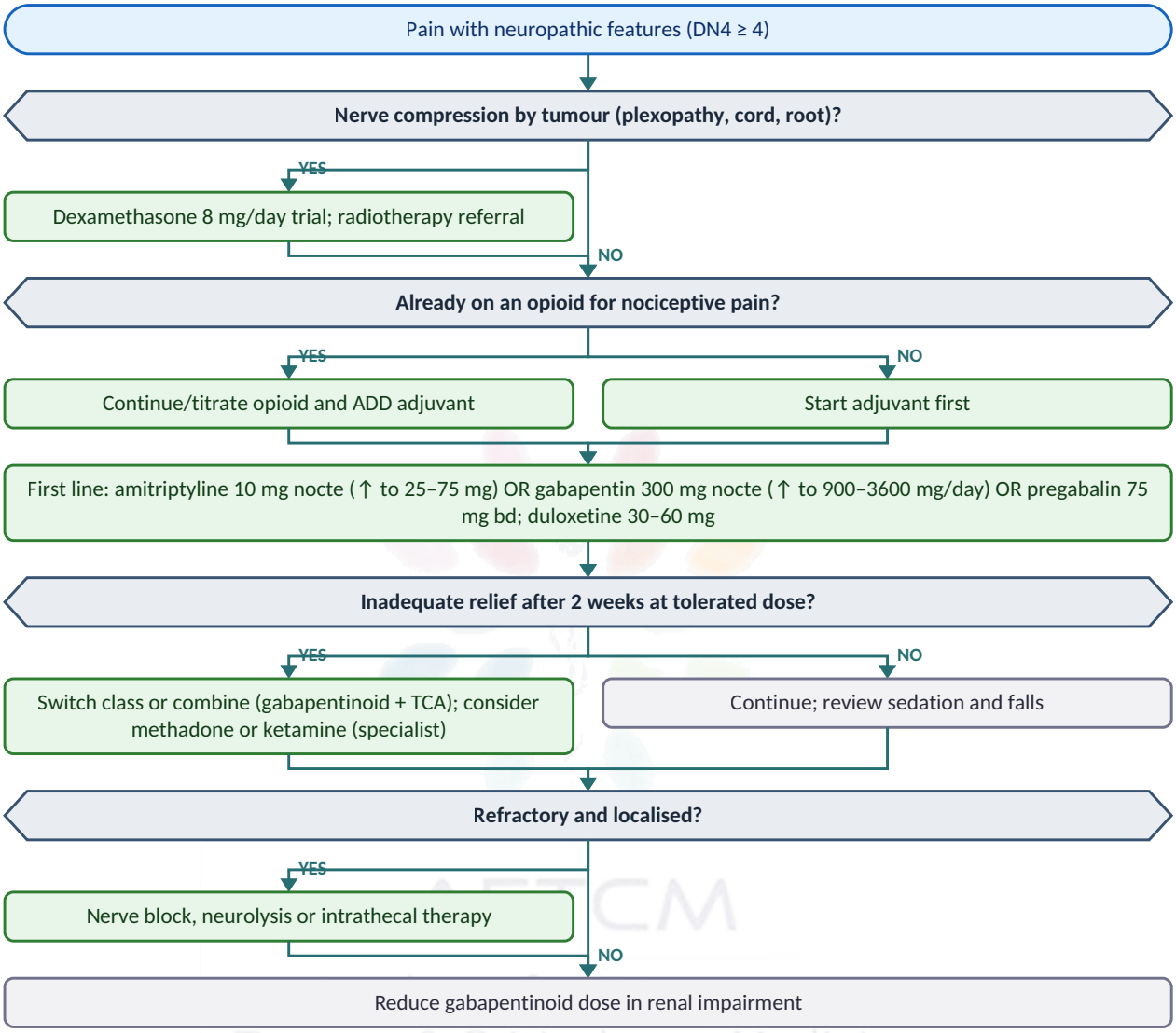
MODEL SHORT-ESSAY ANSWER · SE 5

Pharmacotherapy of Neuropathic Pain

Read the text ›

Neuropathic pain arises from damage or dysfunction of the somatosensory nervous system (e.g. post-mastectomy syndrome, chemotherapy-induced peripheral neuropathy, tumour nerve infiltration), classically described as burning, shooting, or electric-shock-like, often with allodynia. It responds poorly to opioids alone. First-line agents are gabapentinoids (gabapentin, pregabalin) and tricyclic antidepressants (amitriptyline) or SNRIs (duloxetine); second-line/refractory options include ketamine and topical lidocaine/capsaicin for localised pain. A modest opioid may be combined for mixed nociceptive-neuropathic pain, but adjuvants — not opioid escalation — are the mainstay.

ALGORITHM · Neuropathic Pain Pharmacotherapy Algo 25 — full page ›



★ **KEY POINTS**

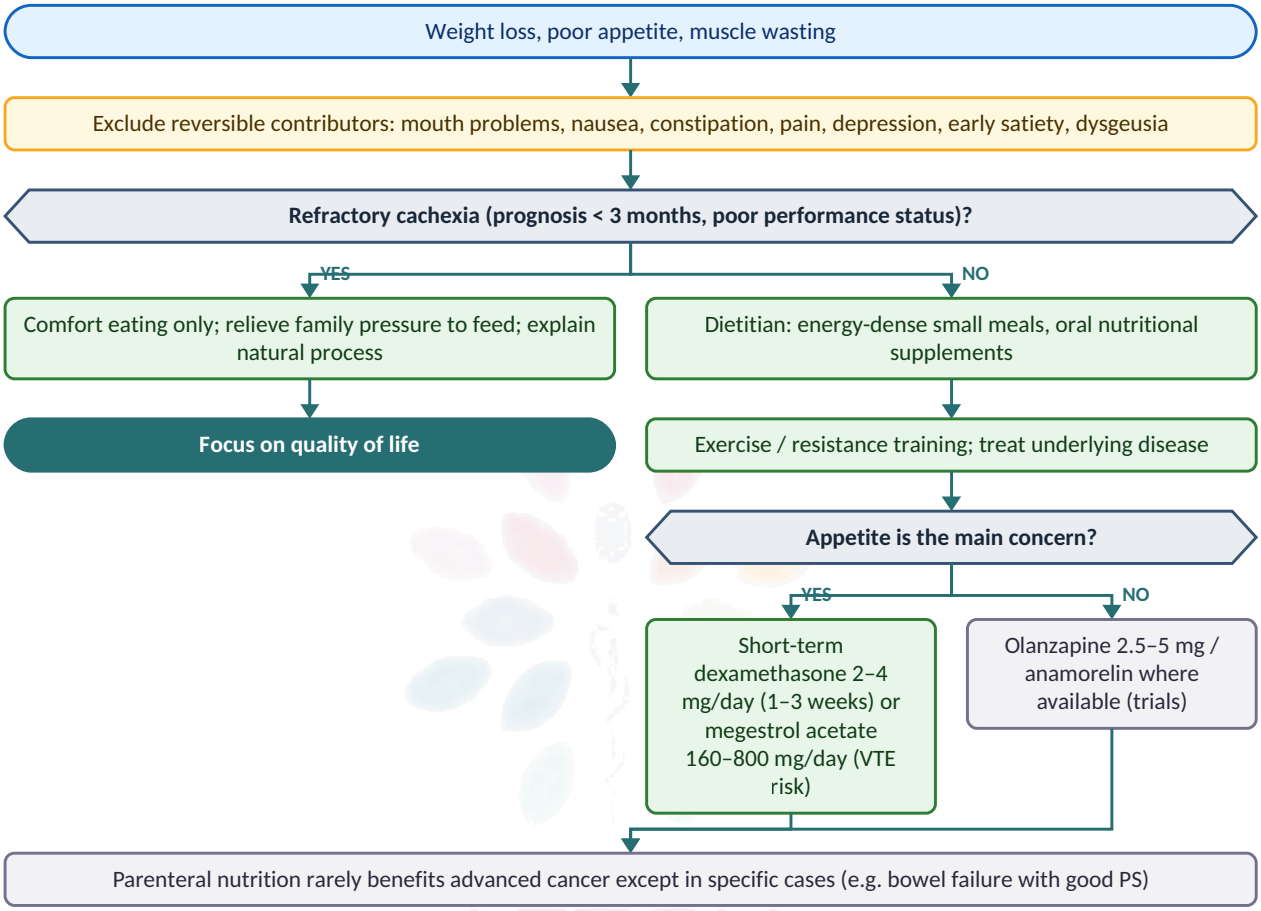
- Number needed to treat ~3–4 for TCAs; ~6–8 for gabapentinoids.
- Amitriptyline: caution in cardiac disease and elderly.

MODEL SHORT-ESSAY ANSWER · SE 6

Cancer Cachexia-Anorexia Syndrome

Cancer cachexia is a multifactorial syndrome of progressive loss of skeletal muscle (with or without fat loss) driven by systemic inflammation and tumour-derived catabolic factors, not simply reduced intake, and is not fully reversible by nutritional support alone. It is associated with anorexia, early satiety, and profound fatigue, and worsens prognosis and treatment tolerance. Management includes treating reversible contributors (mucositis, nausea, constipation, pain, depression), dietary counselling with small frequent meals, and in selected patients a trial of corticosteroids or progestogens (megestrol acetate) for short-term appetite stimulation; aggressive artificial nutrition is generally not beneficial in advanced, irreversible cachexia and may add burden without benefit.

ALGORITHM · Cancer Anorexia–Cachexia Syndrome Algo 40 — full page ›



★ KEY POINTS

- Cachexia is driven by inflammation (IL-6, TNF-α) — not simply starvation.
- Family distress over 'not eating' is a major issue — address it explicitly.

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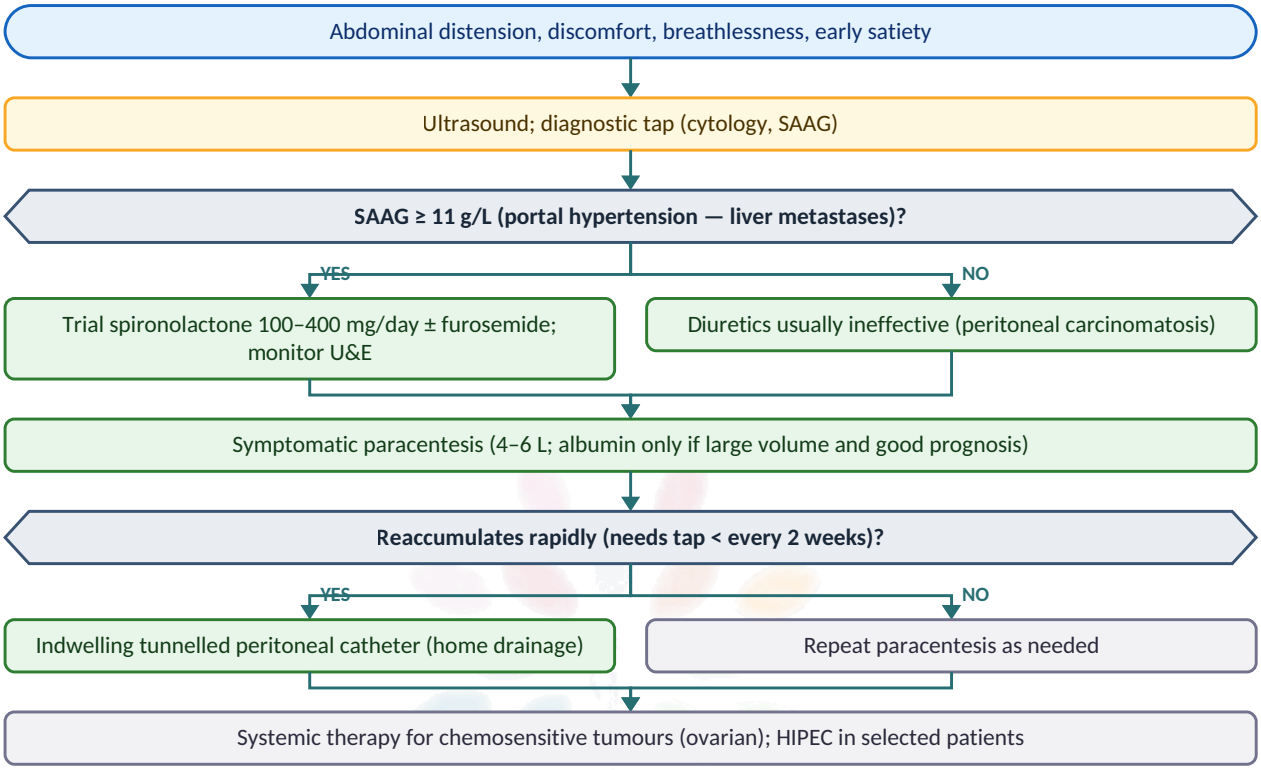
MODEL SHORT-ESSAY ANSWER · SE 7

Malignant Ascites

Read the text ›

Malignant ascites results from peritoneal carcinomatosis (commonly ovarian, gastrointestinal, breast cancers) causing increased peritoneal fluid production and impaired lymphatic drainage, producing abdominal distension, discomfort, breathlessness, and early satiety. Therapeutic paracentesis provides symptomatic relief, though fluid often reaccumulates; a diuretic trial (spironolactone ± furosemide) may help, particularly when a portal-hypertensive component contributes. Recurrent cases may benefit from an indwelling peritoneal catheter for repeated home drainage, improving quality of life by avoiding repeated hospital procedures.

ALGORITHM · Malignant Ascites Algo 41 — full page ›



★ KEY POINTS

- Malignant ascites median survival is often < 6 months — discuss goals.
- Catumaxomab (EpCAM) was used in Europe for EpCAM+ ascites.

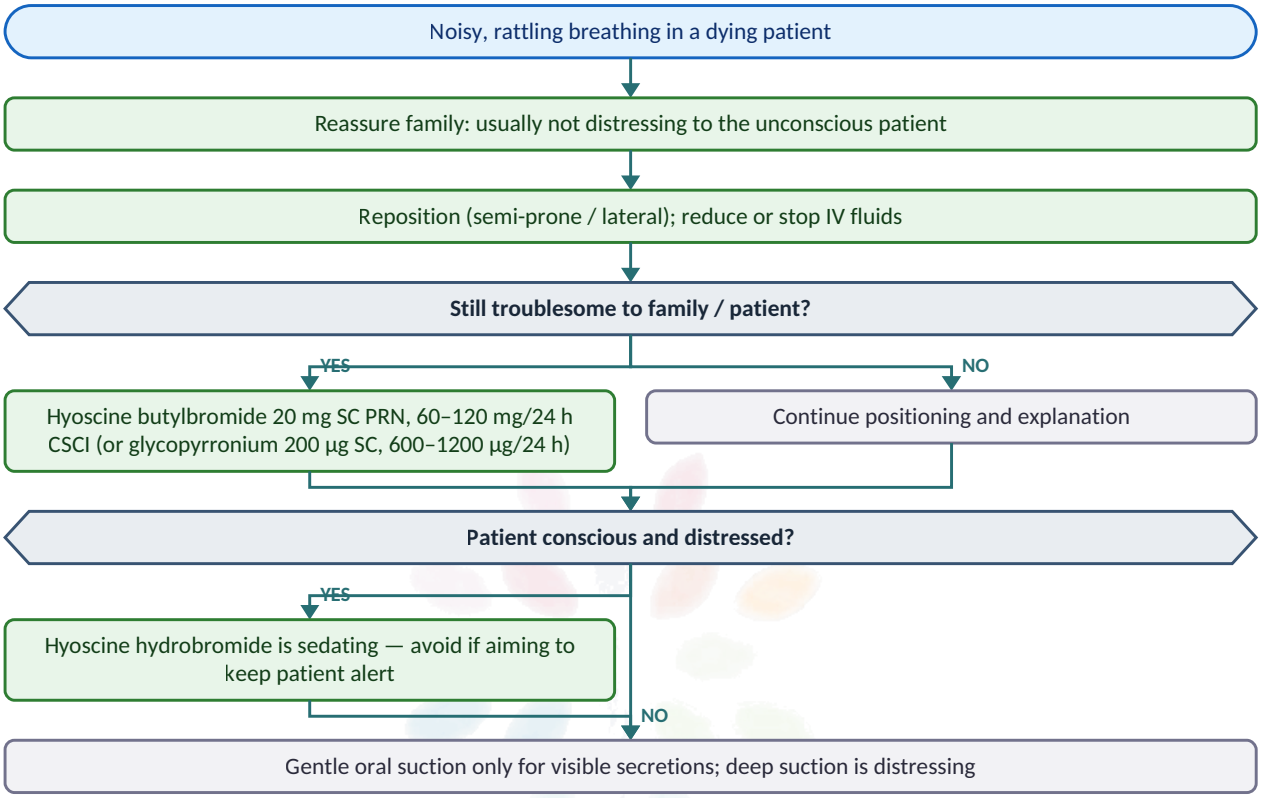
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MODEL SHORT-ESSAY ANSWER · SE 8

Terminal Respiratory Secretions (Death Rattle)

Noisy, rattling breathing from pooled upper-airway secretions in a patient too weak to clear them is common in the last hours of life and, while usually not distressing to the unconscious patient, can be highly distressing to witnessing family. Management is primarily anticholinergic — hyoscine butylbromide, hyoscine hydrobromide, or glycopyrrolate — started early (anticipatory prescribing) as it is more effective at preventing secretion accumulation than clearing established secretions. Repositioning (lateral position) can help drainage. Family should be reassured this is a natural part of dying and not usually a sign of suffering or choking.

ALGORITHM · Terminal Respiratory Secretions (Death Rattle) Algo 42 — full page ›



★ KEY POINTS

- Anticholinergics prevent new secretions but do not clear existing ones — start early.
- Evidence for antimuscarinics is weak (Cochrane) — explanation is the core intervention.

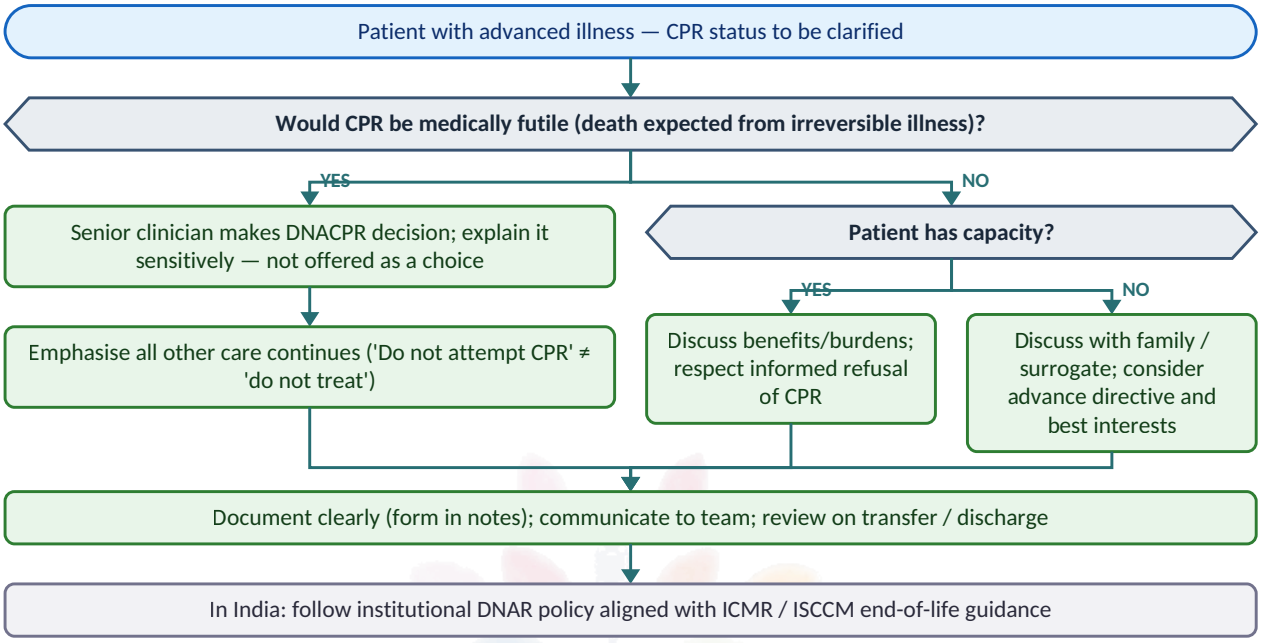
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MODEL SHORT-ESSAY ANSWER · SE 9

Do-Not-Resuscitate (DNR) Orders

A DNR order documents that cardiopulmonary resuscitation should not be attempted if a patient suffers cardiac or respiratory arrest, made when CPR is judged futile or inconsistent with the patient's wishes/goals of care. It should follow a sensitive discussion explaining the rationale, ideally involving the patient (if able) and family, distinguishing clearly that a DNR order applies only to resuscitation and does not mean withdrawal of other active symptom-focused or even disease-directed care. Clear documentation and communication across the care team prevents confusion during an acute event.

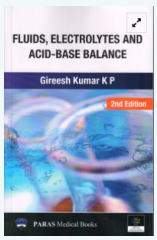
ALGORITHM · Do-Not-Attempt CPR (DNACPR) Decision Algo 43 — full page ›



★ KEY POINTS

- 'Allow natural death' language is better understood by families.
- DNACPR does not mean withholding antibiotics, fluids or symptom control.

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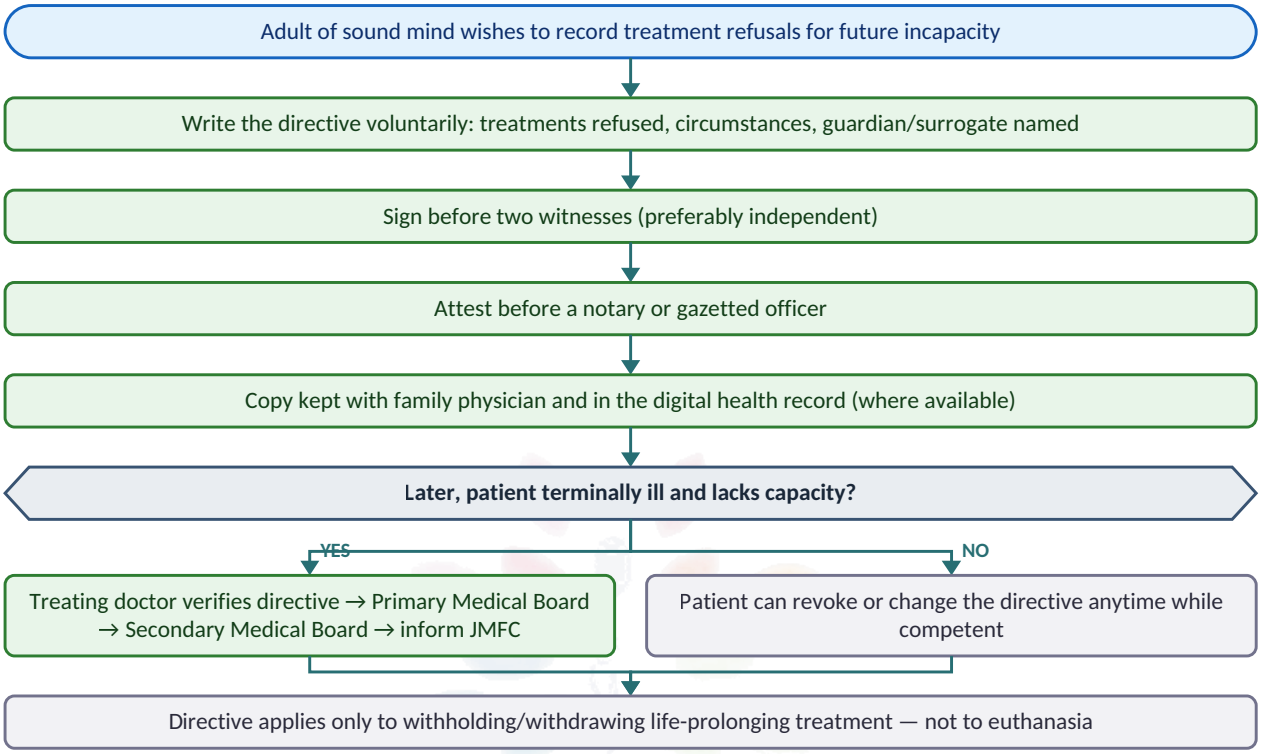
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MODEL SHORT-ESSAY ANSWER · SE 10

Advance Care Directives / Living Will in India

Following Common Cause vs Union of India (2018, procedure simplified 2023), a competent adult in India may execute a written Advance Medical Directive (living will) specifying treatments they would or would not want if they later become incapable of making decisions, including refusal of life-sustaining treatment. The directive must be executed following the prescribed procedure (witnesses, and now a simplified certification process) to be legally actionable. It operationalises patient autonomy into future incapacity and provides legal clarity for families and clinicians facing withdrawal/withholding decisions, though public awareness and uptake in India remain low.

ALGORITHM · Making an Advance Medical Directive in India Algo 44 — full page ›



★ **KEY POINTS**

- Advance care planning conversations matter more than the form itself.
- Name a trusted surrogate who knows the patient's values.

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MODEL SHORT-ESSAY ANSWER · SE 11

Hospice versus Palliative Care

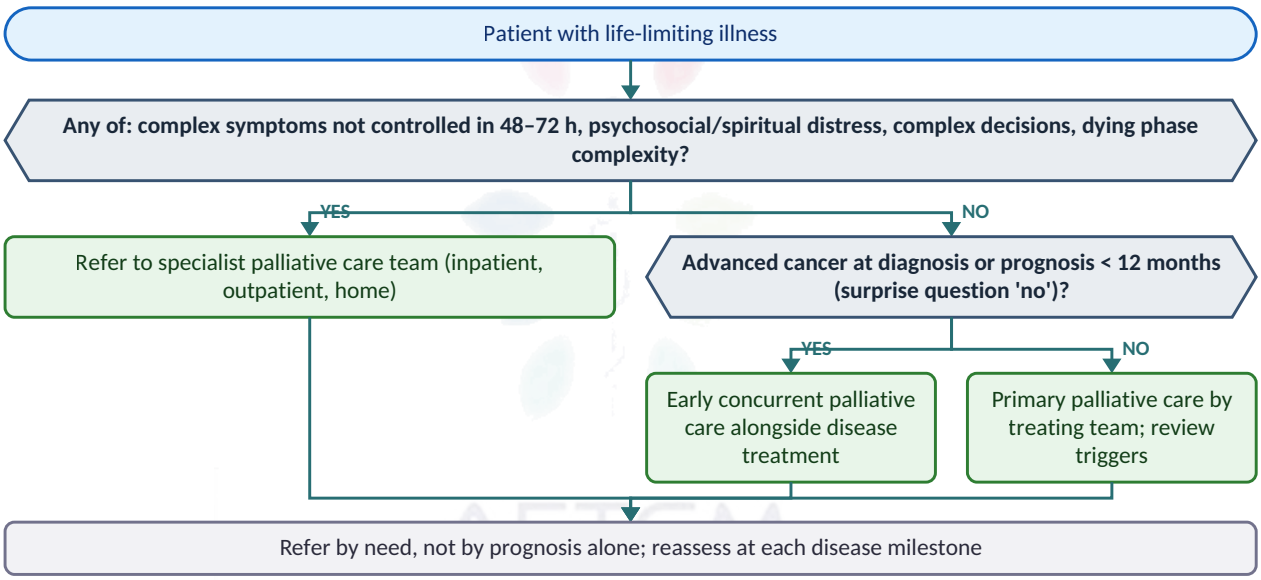
Palliative care is a broad approach to improving quality of life for patients with serious illness, applicable from diagnosis onward, alongside curative or disease-modifying treatment, at any stage and prognosis. Hospice care is a specific model of care, typically for patients with a limited prognosis (often defined as weeks to a few months), focused exclusively on comfort rather than disease-directed treatment, usually delivered at home or in a dedicated facility. All hospice care is palliative care, but not all palliative care is hospice — palliative care has a much wider scope across the illness trajectory, not confined to the terminal phase.

EXAM-ORIENTED THEORY

Hospice care versus palliative care

Feature	Palliative care	Hospice care
Timing	From diagnosis of serious illness, alongside curative treatment	Last months of life (often prognosis < 6 months)
Setting	Hospital, outpatient, home	Hospice unit or home
Disease-directed treatment	Continues	Usually stopped
Focus	Symptom control, QOL, decision support	Comfort and dignity at end of life
Family	Supported throughout	Includes bereavement care

ALGORITHM · When to Refer to Specialist Palliative Care Algo 53 — full page ›



★ KEY POINTS

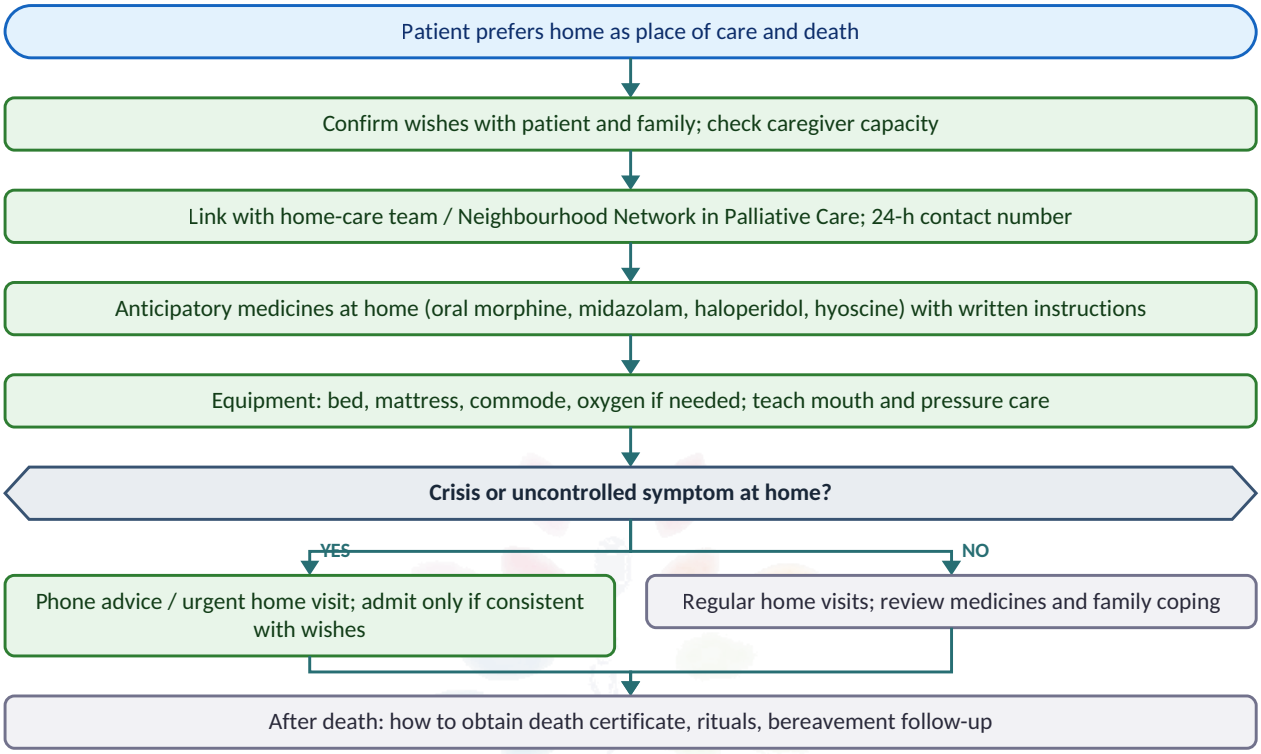
- ASCO recommends specialist palliative referral within 8 weeks of advanced cancer diagnosis.
- Palliative care is not only for the dying.

MODEL SHORT-ESSAY ANSWER · SE 12

Home-Based Palliative Care (the Kerala Model)

Home-based palliative care delivers assessment, symptom management, and psychosocial-spiritual support to patients in their own homes, particularly valuable in India given limited inpatient hospice capacity and strong patient preference to remain at home. Kerala's Neighbourhood Network in Palliative Care pioneered a community-owned model in which trained local volunteers identify patients, provide practical and emotional support, and link them to professional clinical teams for medical needs — achieving substantially higher population coverage than the Indian national average and serving as a widely cited, scalable, low-cost model for community-driven palliative care.

ALGORITHM · Planning for Death at Home Algo 61 — full page ›



★ **KEY POINTS**

- Kerala's community-owned model (NNPC) covers a large share of patients at home through volunteers.
- Narcotic Drugs and Psychotropic Substances Amendment Act 2014 eased morphine access.

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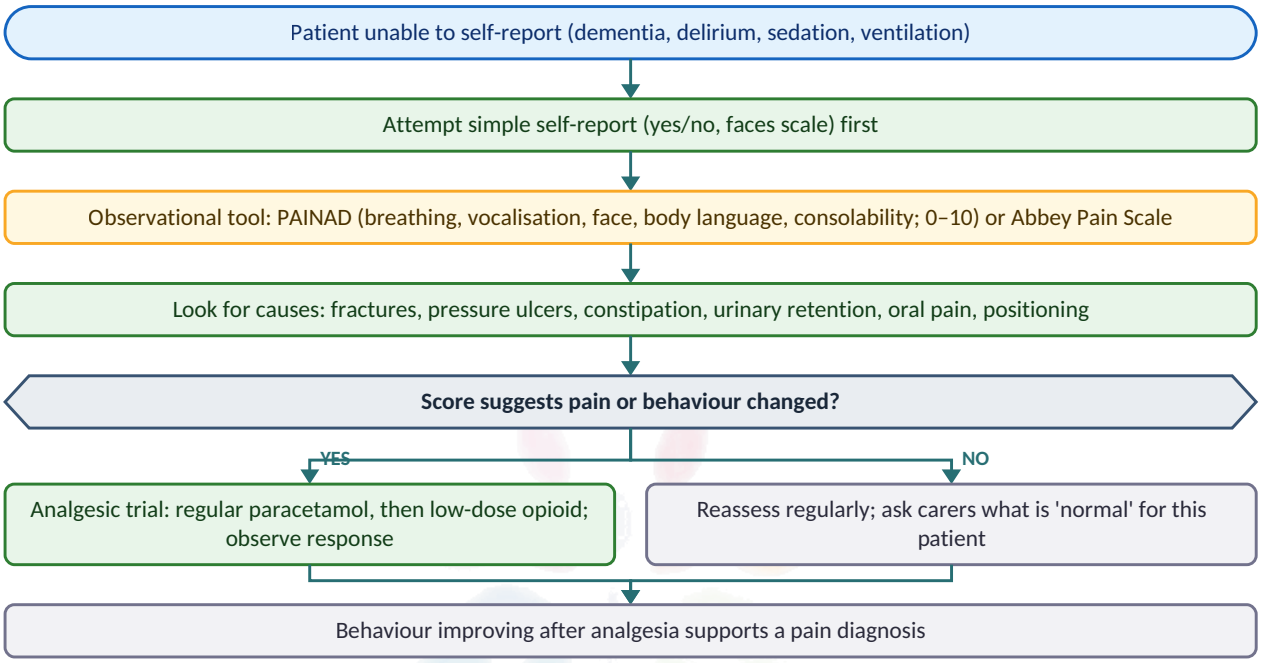
MODEL SHORT-ESSAY ANSWER · SE 13

Pain Assessment in Non-Verbal / Cognitively Impaired Patients

Read the text ›

Self-report, the gold standard for pain assessment, is unreliable or impossible in non-verbal patients (advanced dementia, severe neurological impairment, young infants, or critically ill sedated/ventilated patients). Validated observational/behavioural tools are used instead: PAINAD for advanced dementia (breathing, negative vocalisation, facial expression, body language, consolability), FLACC for young children (Face, Legs, Activity, Cry, Consolability), and CPOT for critically ill/ventilated adults. These tools score observable behavioural and physiological indicators as a proxy for pain intensity and should be used serially to track response to analgesia.

ALGORITHM · Pain Assessment in Non-Verbal and Cognitively Impaired Patients Algo 45 — full page ›



★ KEY POINTS

- Agitation in dementia is often unrecognised pain.
- Critical-care Pain Observation Tool (CPOT) for ventilated patients.

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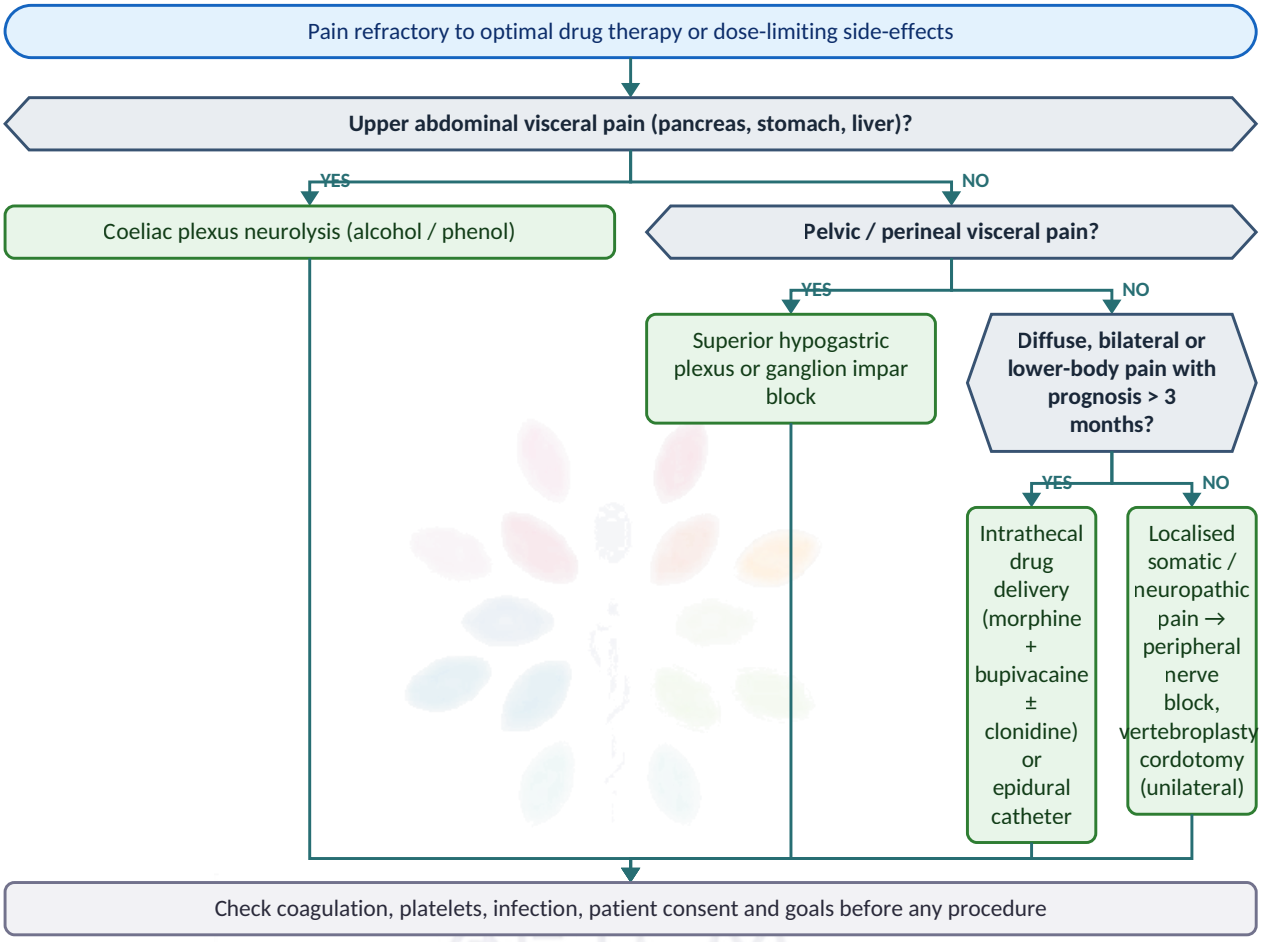
MODEL SHORT-ESSAY ANSWER · SE 14

Interventional Pain Techniques — Overview

Read the text ›

Interventional techniques are reserved for pain refractory to optimised systemic pharmacotherapy or when systemic adverse effects limit further escalation. Options include peripheral/sympathetic nerve blocks (e.g. coeliac plexus block for pancreatic cancer pain, using local anaesthetic or neurolytic agents such as alcohol/ phenol for longer-duration relief), neuraxial (epidural/intrathecal) analgesia delivering opioid ± local anaesthetic close to the spinal cord at much lower systemic doses, and radiofrequency ablation or cordotomy in highly selected refractory cases. These are sometimes described as a conceptual 'fourth step' beyond the WHO ladder, requiring specialist expertise and careful patient selection.

ALGORITHM · Choosing an Interventional Pain Technique Algo 46 — full page ›



★ KEY POINTS

- Early coeliac block reduces opioid use and constipation in pancreatic cancer.
- Percutaneous cervical cordotomy suits unilateral mesothelioma pain.

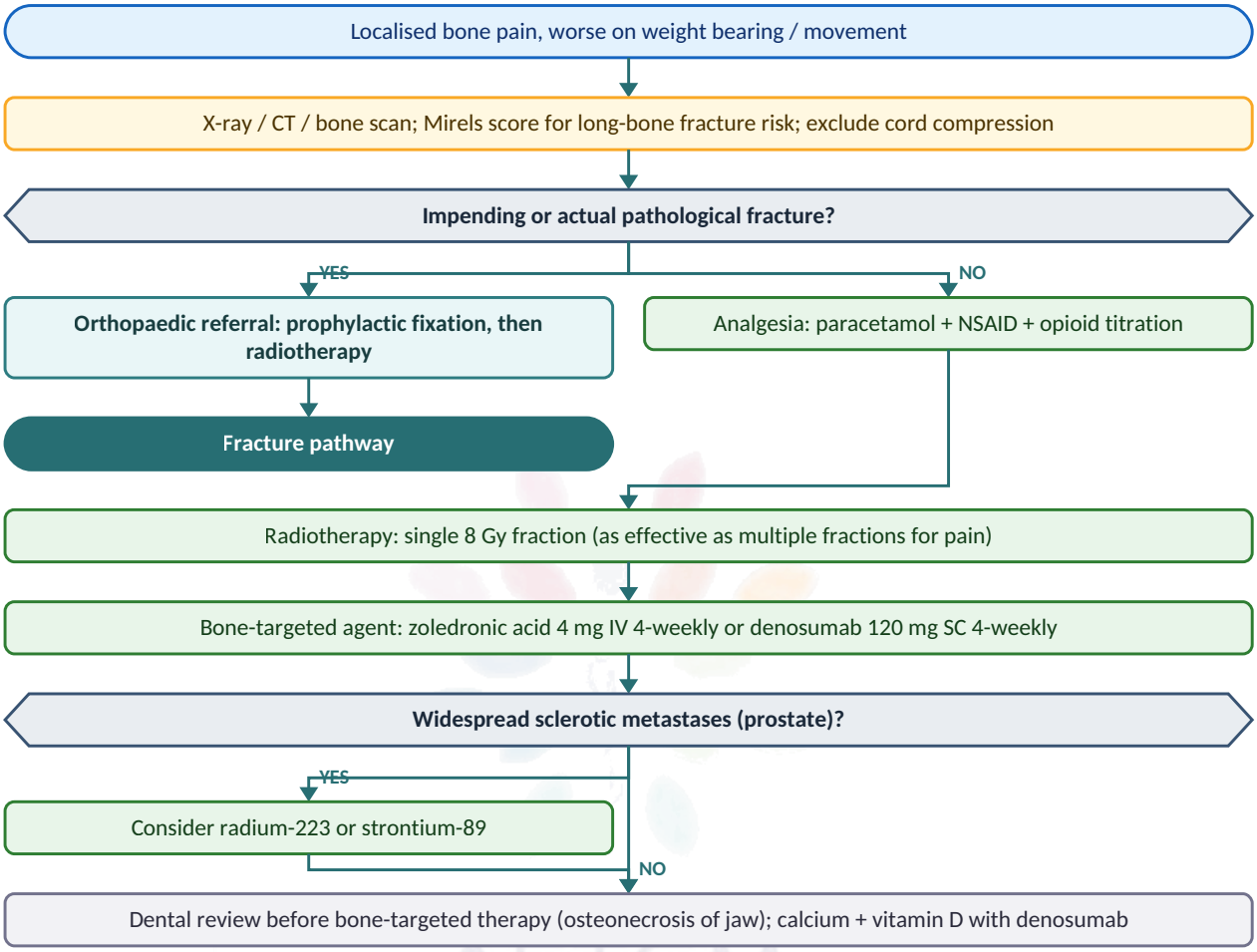
MODEL SHORT-ESSAY ANSWER · SE 15

Radiotherapy for Bone Pain

Read the text ›

External beam radiotherapy is highly effective for localised painful bone metastases, providing meaningful relief in the majority of patients, typically within 1–4 weeks. For uncomplicated bone metastases, a single 8 Gy fraction gives pain relief equivalent to longer multi-fraction regimens (e.g. 20 Gy/5 fractions, 30 Gy/10 fractions), offering a resource-efficient, low-burden option especially valuable in palliative and resource-limited settings, though with a somewhat higher re-treatment rate than fractionated courses. Multi-fraction regimens may be preferred for complicated metastases (impending fracture, existing cord compression) requiring a more durable, higher cumulative dose.

ALGORITHM · Pain from Bone Metastases Algo 26 — full page ›



★ KEY POINTS

- Pain relief after radiotherapy takes 2–4 weeks; pain flare in the first days — cover with dexamethasone.
- Mirels score ≥ 9 → prophylactic fixation.

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MODEL SHORT-ESSAY ANSWER · SE 16

Bisphosphonates and Denosumab in Malignancy

Read the text ›

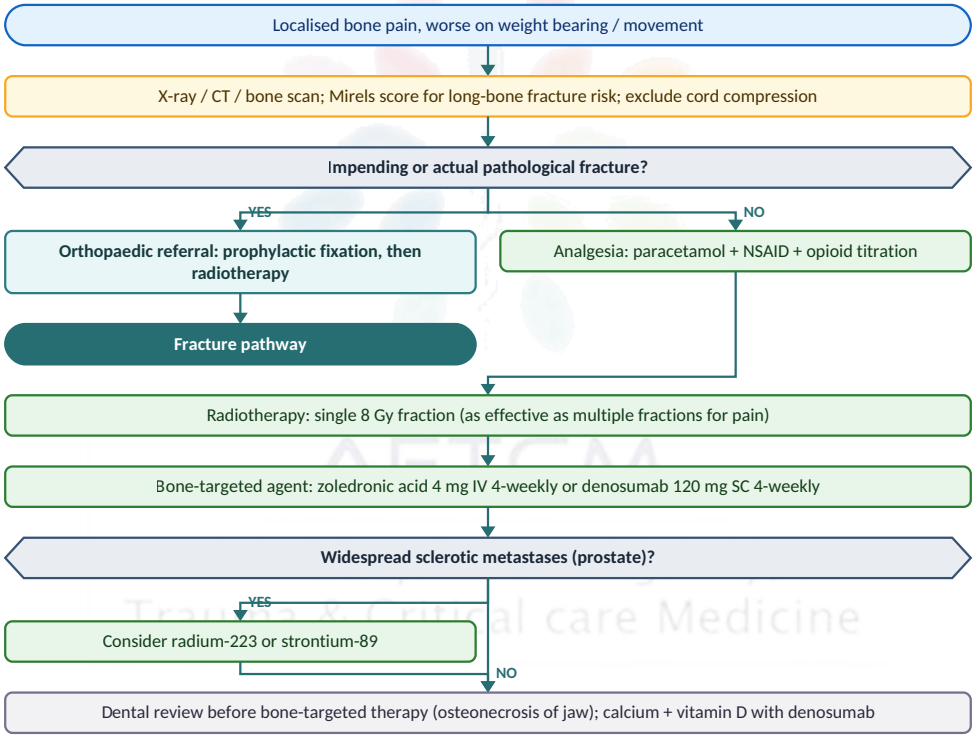
Bisphosphonates (e.g. zoledronic acid) and denosumab (a monoclonal antibody against RANK ligand) are bone-targeted agents that reduce osteoclast-mediated bone resorption, used to prevent skeletal-related events (pathological fracture, cord compression, need for radiotherapy/surgery) in bone metastases and to treat hypercalcaemia of malignancy (bisphosphonates). Both carry a risk of osteonecrosis of the jaw (requiring dental assessment before starting) and hypocalcaemia; bisphosphonates additionally require renal function monitoring and dose adjustment, while denosumab does not require renal dose adjustment but carries a higher rebound hypercalcaemia/fracture risk on discontinuation.

EXAM-ORIENTED THEORY

Zoledronic acid vs denosumab

Feature	Zoledronic acid	Denosumab
Mechanism	Bisphosphonate — osteoclast apoptosis	RANKL monoclonal antibody
Dose	4 mg IV over 15 min every 3–4 weeks (12-weekly option)	120 mg SC every 4 weeks
Renal impairment	Dose-adjust; avoid if CrCl < 30	No adjustment; higher hypocalcaemia risk
Acute-phase reaction	Common with first dose	Rare
Osteonecrosis of jaw	1–2%	1–2% (slightly higher)
Rebound on stopping	No	Yes — rebound vertebral fractures
Evidence	Delays skeletal-related events	Superior delay of SREs (Stopeck 2010)

ALGORITHM · Pain from Bone Metastases Algo 26 — full page ›



★ KEY POINTS

- Pain relief after radiotherapy takes 2–4 weeks; pain flare in the first days — cover with dexamethasone.
- Mirels score ≥ 9 → prophylactic fixation.

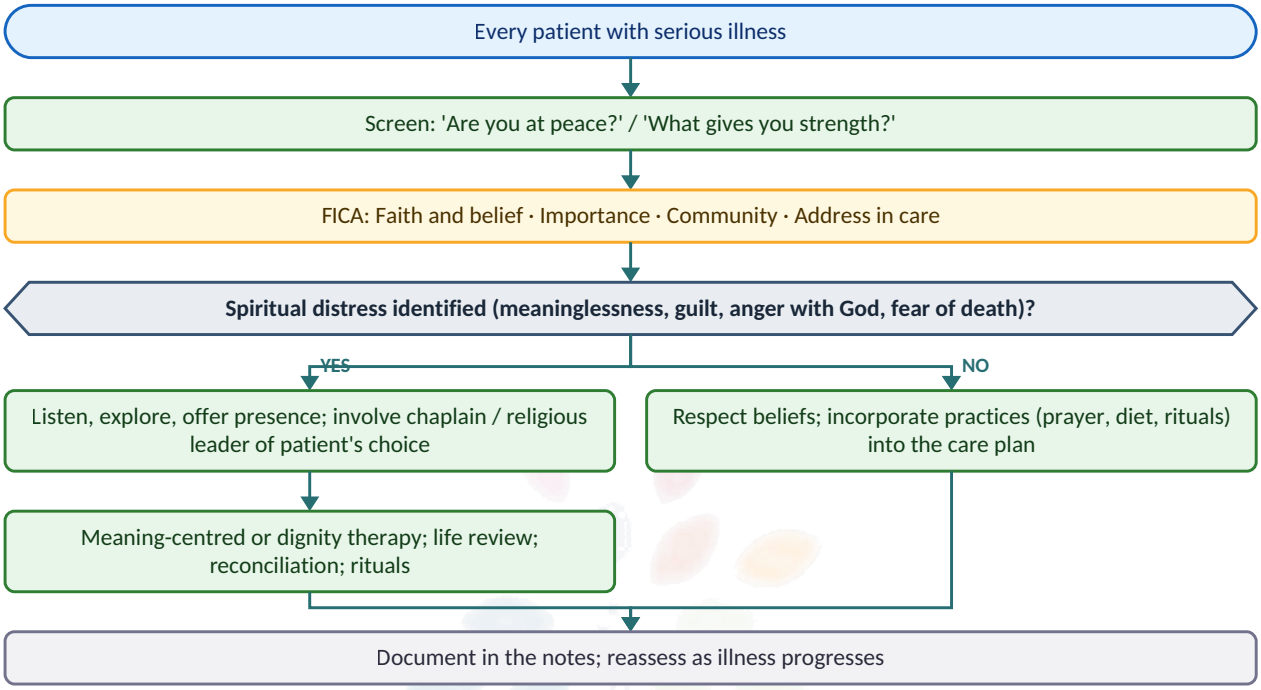
MODEL SHORT-ESSAY ANSWER · SE 17

Spiritual Distress and Assessment

Read the text ›

Spiritual distress refers to suffering in the existential domain — loss of meaning, hope, or connection, unresolved guilt, anger at a higher power, or fear of death — and is a recognised component of Cicely Saunders' total pain concept, independent of religious affiliation. Structured tools such as FICA (Faith, Importance, Community, Address in care) and HOPE (sources of Hope, Organised religion, Personal spirituality, Effects on care) allow routine, non-specialist eliciting of spiritual needs. Simple interventions — unhurried presence, facilitating religious rituals, reconciliation, and legacy work — can substantially ease spiritual suffering, with specialist chaplaincy referral for deeper needs.

ALGORITHM · Spiritual Assessment (FICA) and Care Algo 39 — full page ›



★ **KEY POINTS**

- Spirituality is broader than religion — it is about meaning and connection.
- HOPE and SPIRIT are alternative assessment tools.

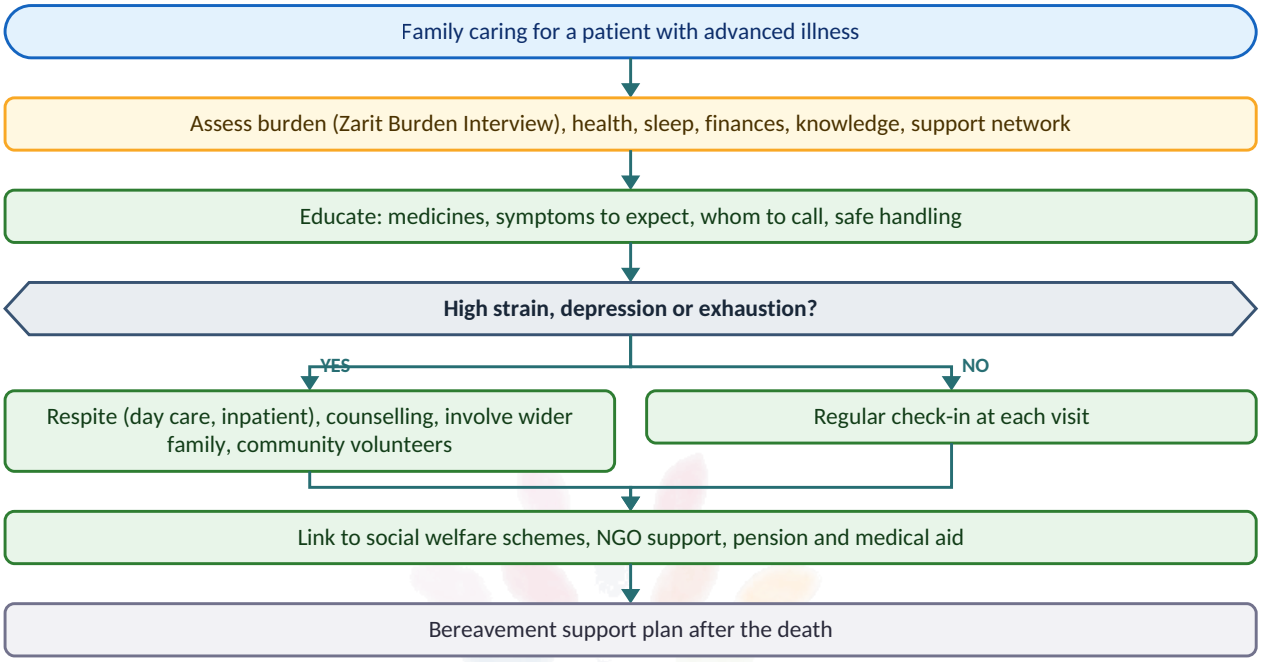
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MODEL SHORT-ESSAY ANSWER · SE 18

Caregiver Burden and Burnout

Family caregivers of patients with advanced illness frequently experience significant physical, emotional, financial, and social strain — caregiver burden — which, left unaddressed, predisposes to caregiver burnout (exhaustion, depression, resentment) and poorer bereavement outcomes. Palliative teams should proactively screen caregivers for burden (not only focus on the patient), provide respite options where available, practical skills training (e.g. safe moving/handling, medication administration), psychosocial support, and connect families to financial/social support resources, recognising the caregiver as a co-recipient of care, not merely a care provider.

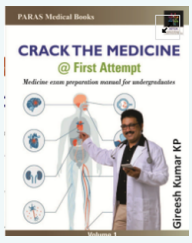
ALGORITHM · Supporting the Family Caregiver Algo 47 — full page ›



★ KEY POINTS

- Caregiver burden predicts complicated grief.
- Ask the caregiver 'How are you coping?' at every visit.

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MODEL SHORT-ESSAY ANSWER · SE 19

Compassion Fatigue in Palliative Care Providers

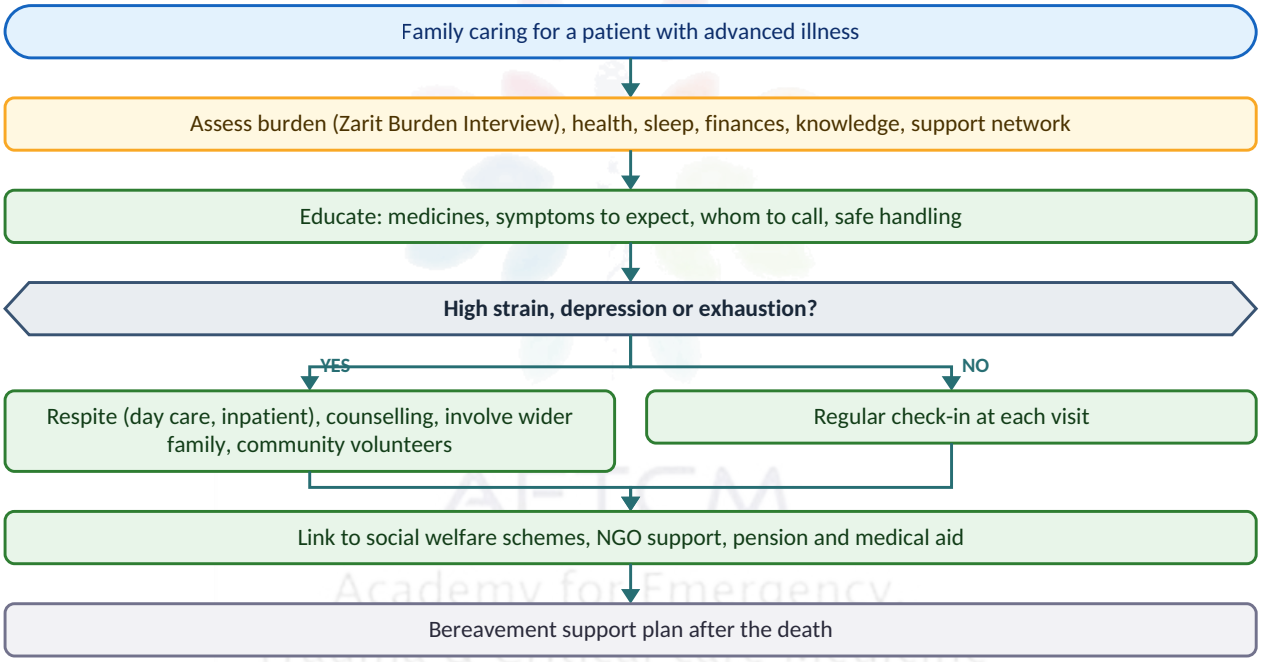
Compassion fatigue is the cumulative emotional and physical exhaustion experienced by healthcare providers from sustained empathetic engagement with suffering and dying patients, related to but distinct from general burnout, and a recognised occupational hazard in palliative medicine. Features include emotional exhaustion, reduced empathy, cynicism, and physical symptoms. Prevention and management strategies include regular debriefing and peer support, manageable caseloads, mindfulness/self-care practices, institutional recognition and support structures, and seeking professional help when symptoms are significant — essential not only for provider wellbeing but for sustaining quality patient care.

EXAM-ORIENTED THEORY

Preventing compassion fatigue and burnout

Level	Strategy
Individual	Self-awareness, reflective practice, mindfulness, boundaries, leave, exercise, peer support
Team	Debriefing after difficult deaths, Schwartz rounds, shared workload, recognition
Organisation	Adequate staffing, supervision, training in communication, psychological services
Warning signs	Emotional exhaustion, cynicism/depersonalisation, reduced accomplishment (Maslach Burnout Inventory)
Compassion satisfaction	Meaning from the work — protective; measure with ProQOL

ALGORITHM · Supporting the Family Caregiver Algo 47 — full page ›



★ KEY POINTS

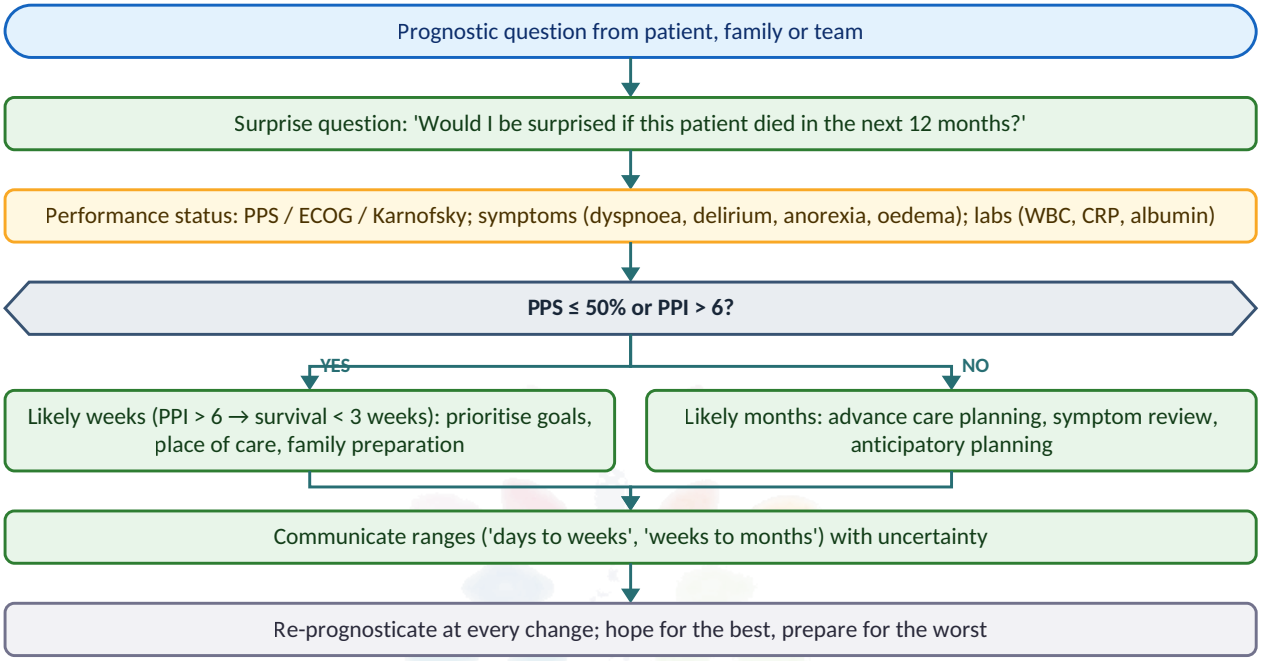
- Caregiver burden predicts complicated grief.
- Ask the caregiver 'How are you coping?' at every visit.

MODEL SHORT-ESSAY ANSWER · SE 20

Prognostication in Advanced Cancer

Accurate prognostication guides treatment decisions, discharge planning, and the timing of difficult conversations, though clinician estimates are frequently overoptimistic. Clinical tools include the Palliative Performance Scale and Karnofsky Performance Status (functional decline correlates with shortened survival), and composite prognostic scores such as the Palliative Prognostic Index and Palliative Prognostic Score, which combine performance status, symptoms (dyspnoea, anorexia), and simple laboratory markers. Clinical signs suggesting a prognosis of days (the actively dying phase) include profound weakness, minimal oral intake, altered consciousness, and characteristic breathing pattern changes. Honest, appropriately caveated prognostic communication supports informed patient and family decision-making.

ALGORITHM · Prognostication in Advanced Illness Algo 48 — full page ›



★ KEY POINTS

- PaP score uses dyspnoea, anorexia, KPS, CPS, WBC and lymphocyte %.
- Rate of change in function is a powerful prognostic clue.

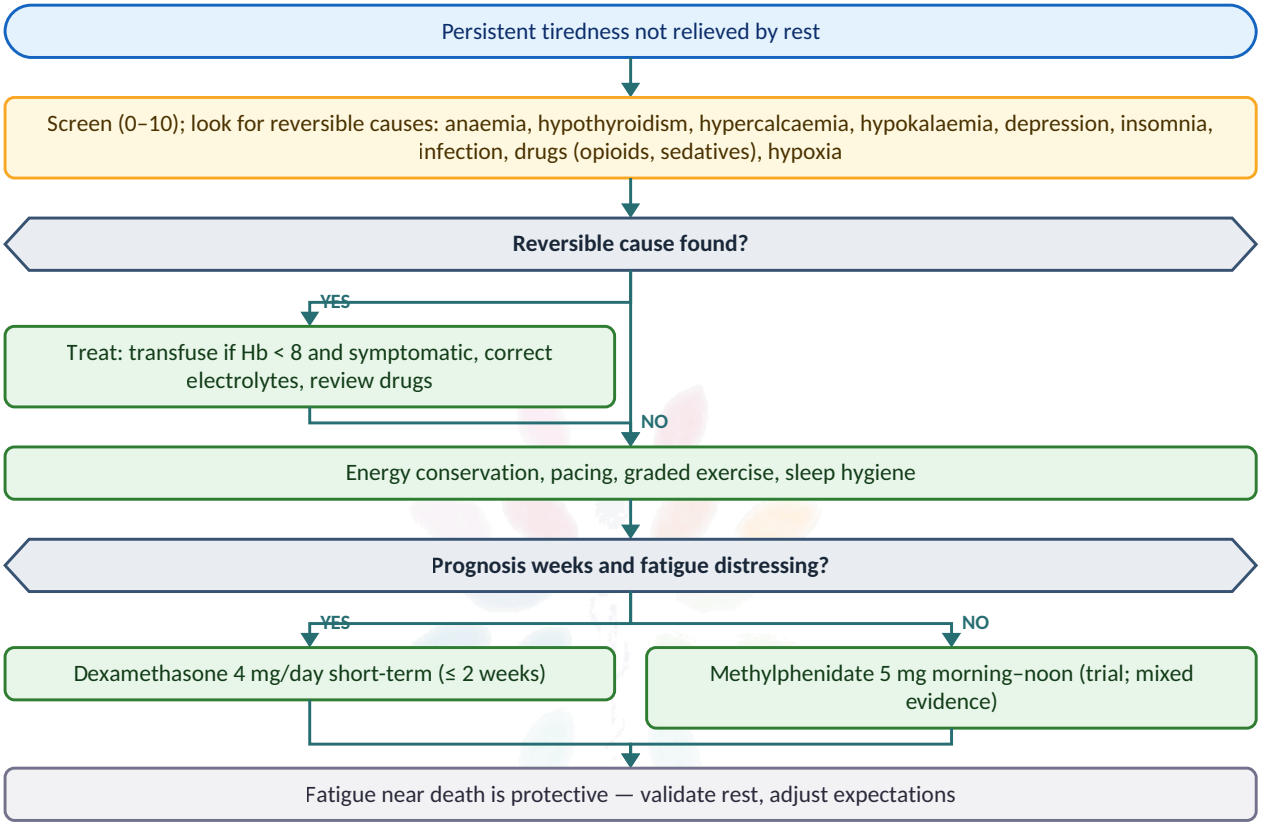
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MODEL SHORT-ESSAY ANSWER · SE 21

Fatigue in Palliative Care

Fatigue is among the most prevalent and distressing symptoms in advanced illness, often under-recognised and undertreated relative to pain. Contributors include the disease process itself (cancer-related fatigue, cytokine-mediated), anaemia, poor sleep, deconditioning, medication effects (opioids, sedatives), depression, and metabolic/electrolyte disturbance. Management begins with identifying and treating reversible contributors (correcting anaemia if appropriate to goals of care, reviewing sedating medications, treating depression); non-pharmacological measures (graded exercise/physiotherapy where feasible, energy conservation techniques, sleep hygiene) have the best evidence; psychostimulants (e.g. methylphenidate) are sometimes used in selected refractory cases with modest evidence.

ALGORITHM · Fatigue in Palliative Care Algo 49 — full page ›



★ KEY POINTS

- Exercise has the strongest evidence for cancer-related fatigue in earlier disease.
- Steroid benefit wears off after 2–4 weeks.

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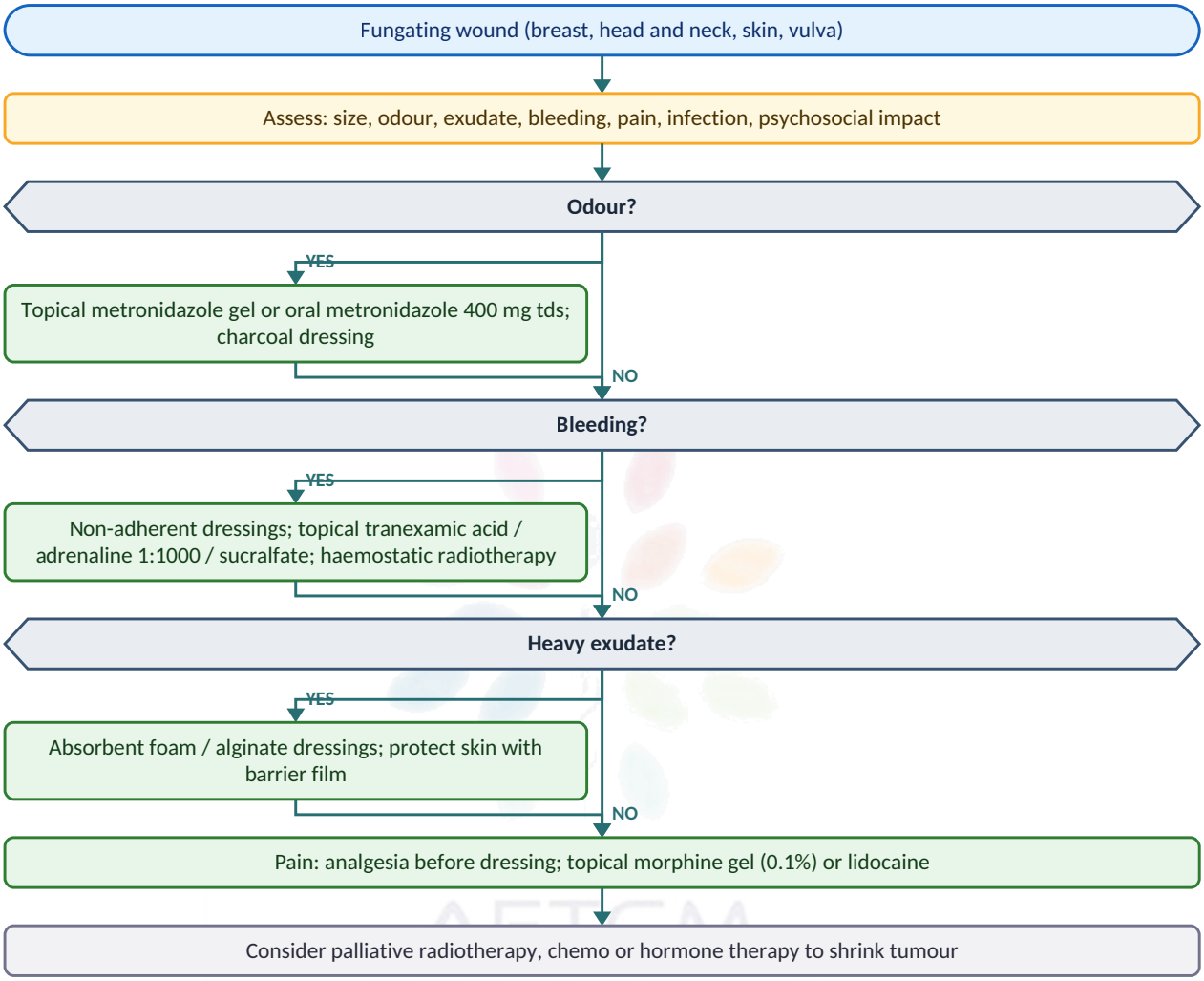
MODEL SHORT-ESSAY ANSWER · SE 22

Malignant Wound Care

Read the text ›

Malignant (fungating) wounds result from direct tumour infiltration of skin and underlying tissue, causing exudate, malodour, bleeding, and pain, with significant psychosocial impact from visible disfigurement and odour. Management focuses on symptom control rather than healing: appropriate absorptive dressings for exudate, topical/systemic metronidazole for anaerobic odour control, careful non-adherent dressing changes and topical sucralfate or adrenaline-soaked gauze for bleeding, adequate analgesia before dressing changes (anticipating incident pain), and charcoal dressings for odour. Psychological support addressing body image and social isolation is equally important.

ALGORITHM · Malignant Fungating Wound Care Algo 50 — full page ›



★ KEY POINTS

- Odour is the most distressing feature for patients and families.
- Change dressings gently — irrigate rather than scrub.



ILLUSTRATION

Dressing materials for a fungating wound

- Non-adherent contact layer, with foam or alginate to absorb exudate.
- Odour: charcoal dressing plus topical metronidazole.
- Bleeding: alginate, gentle pressure, topical tranexamic acid.
- Give analgesia before dressing changes; irrigate gently with saline — never scrub.

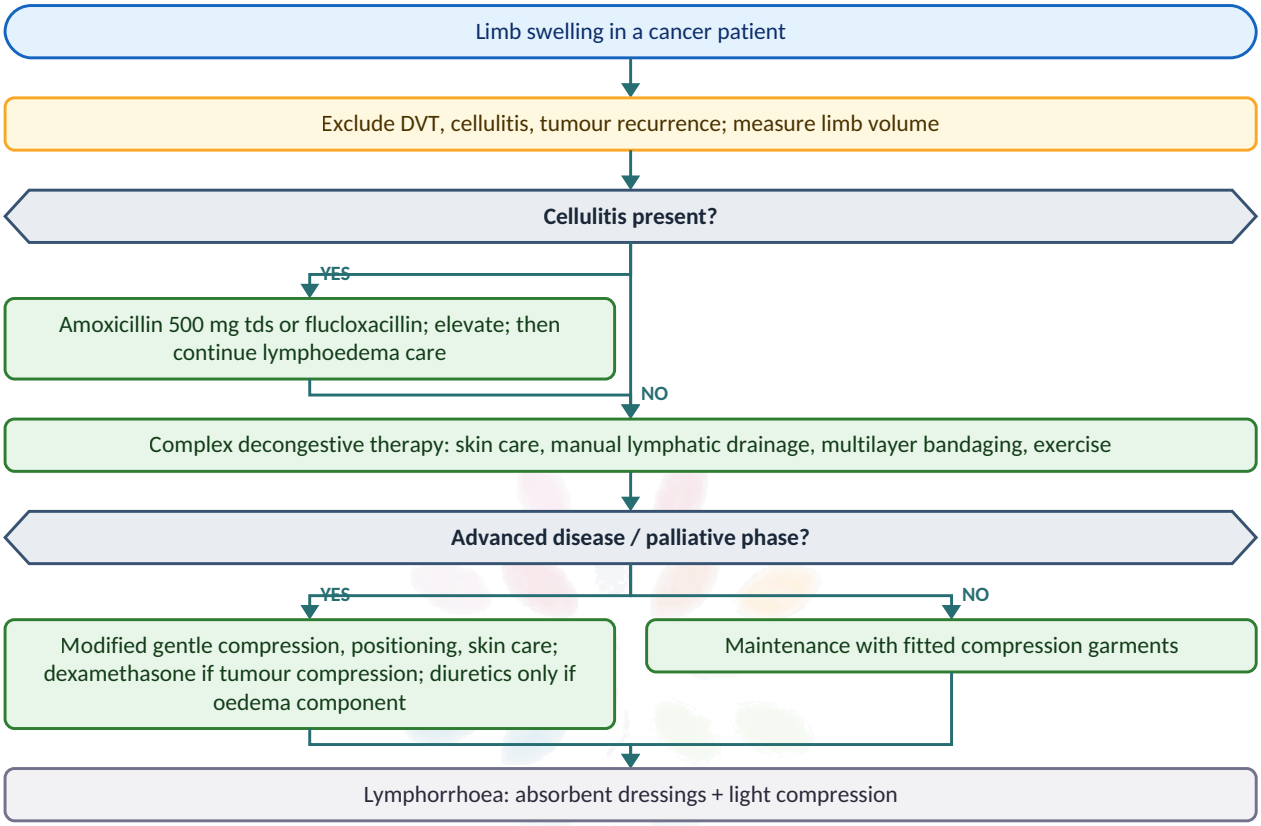
MODEL SHORT-ESSAY ANSWER · SE 23

Lymphedema Management

Read the text ›

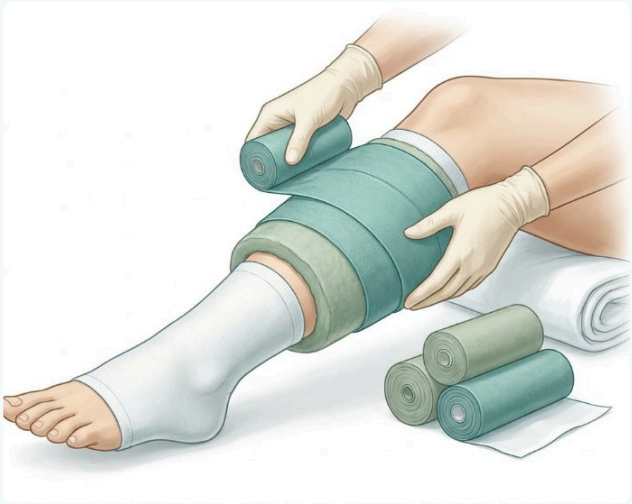
Lymphedema results from lymphatic obstruction by tumour or treatment (lymph node dissection, radiotherapy), causing chronic limb swelling, heaviness, discomfort, and increased infection (cellulitis) risk. Management follows complete decongestive therapy: manual lymphatic drainage, multilayer compression bandaging followed by maintenance compression garments, skin care to reduce infection risk, and remedial exercises; elevation provides modest adjunctive benefit. Diuretics are generally ineffective for lymphedema (protein-rich fluid) and not routinely recommended. Early referral to a trained lymphedema therapist improves outcomes.

ALGORITHM · Lymphoedema Management Algo 51 — full page ›



★ KEY POINTS

- Lymphoedema does not respond to diuretics.
- Skin care prevents cellulitis — the commonest complication.



ILLUSTRATION

Multilayer lymphoedema bandaging

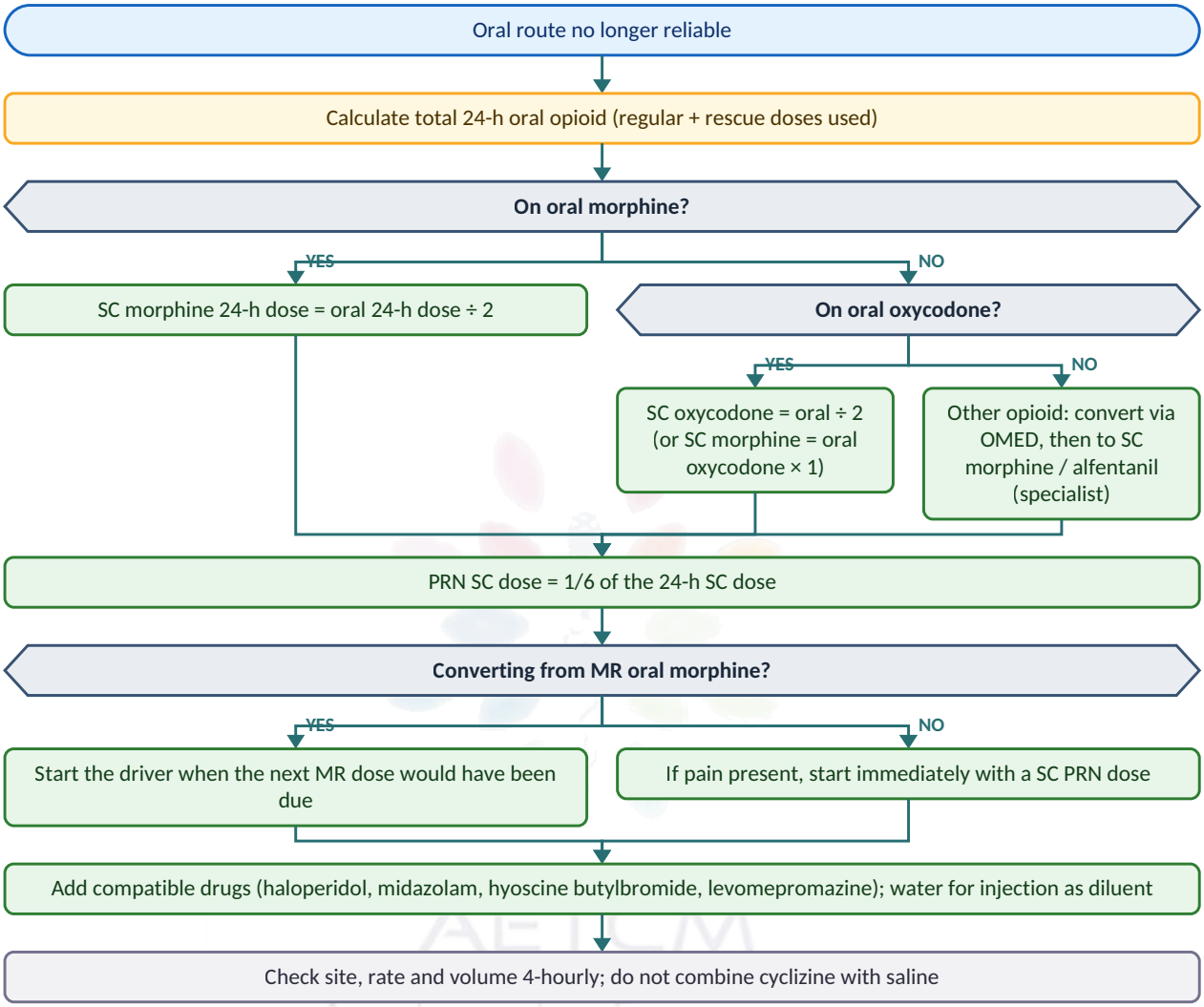
- Part of complex decongestive therapy: skin care, manual lymphatic drainage, compression bandaging, exercise.
- Layers: tubular stockinette → soft padding → short-stretch bandages applied distal to proximal, with graduated pressure.
- Exclude cellulitis and DVT before compressing; remove at once if there is pain, numbness or colour change.
- Once the limb is reduced, change to a fitted compression garment; in advanced disease use lighter bandaging for comfort.

MODEL SHORT-ESSAY ANSWER · SE 24

Syringe Driver / Continuous Subcutaneous Infusion

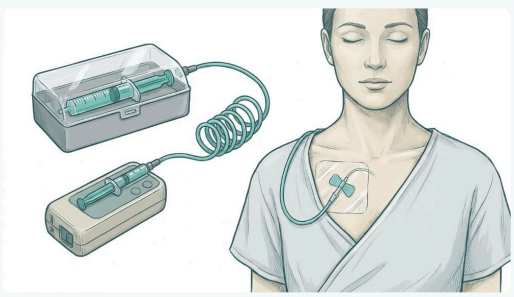
A syringe driver delivers continuous subcutaneous infusion of medication, used when the oral route is no longer feasible (dysphagia, vomiting, reduced consciousness in the dying phase) or when multiple symptoms require combined, steady drug delivery. Commonly combined drugs include an opioid (analgesia), an antiemetic (e.g. haloperidol or cyclizine), an anticholinergic for secretions (hyoscine butylbromide), and a benzodiazepine (midazolam) for agitation — checked for compatibility before mixing in one syringe. The site is checked regularly for erythema/induration and rotated as needed; this route is standard anticipatory-prescribing practice in the last days of life.

ALGORITHM · Converting to a Syringe Driver (CSCI) Algo 28 — full page ›



★ **KEY POINTS**

- Fentanyl patches should be continued, not converted, at end of life — add CSCI for extra needs.
- Driver takes ~4 h to reach steady state.



ILLUSTRATION

Syringe driver (CSCI) set-up

- Site: upper chest, upper arm, abdomen or thigh — avoid oedema, bony and irradiated skin.
- Water for injection as the usual diluent; check drug compatibility.
- Check the site, rate and volume 4-hourly; breakthrough SC dose = 1/6 of the 24-h SC dose.

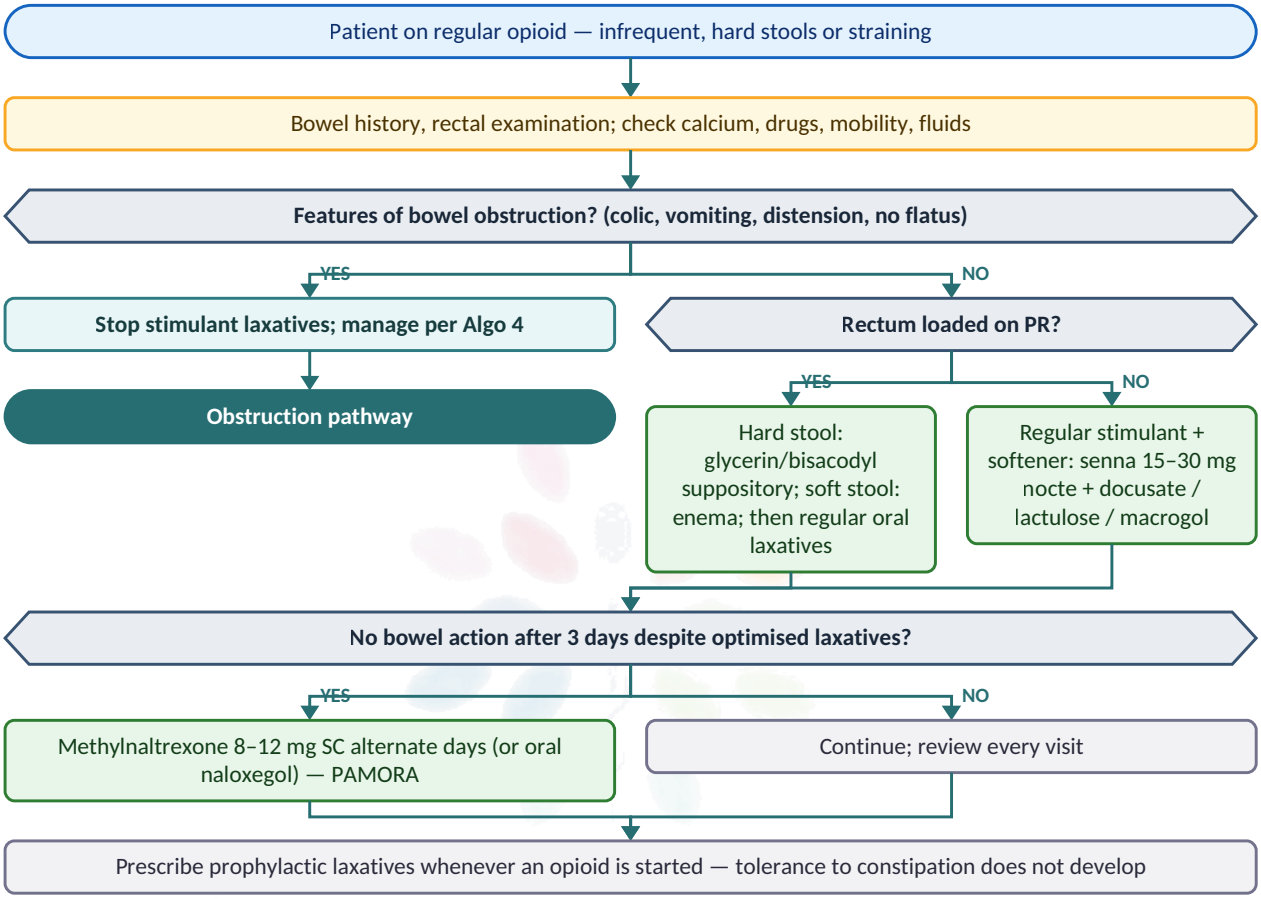
MODEL SHORT-ESSAY ANSWER · SE 25

Opioid-Induced Constipation

Read the text ›

Opioids reduce gastrointestinal motility and secretion via gut mu-receptors, causing constipation in the majority of patients on regular opioids — tolerance does not develop to this effect, unlike sedation or nausea, so prophylaxis is required for as long as the opioid is used. A stimulant laxative (senna) combined with a softener (docusate) is started prophylactically at the time of opioid initiation and titrated to effect; bulk-forming laxatives are generally avoided (risk of worsening obstruction with inadequate fluid intake). For cases refractory to optimised oral laxatives, a peripherally-acting mu-opioid receptor antagonist (e.g. methylnaltrexone) can be used, acting selectively on peripheral gut receptors without reversing central analgesia.

ALGORITHM · Approach to Opioid-Induced Constipation Algo 13 — full page ›



★ KEY POINTS

- PAMORAs are contraindicated in known or suspected obstruction.
- Lactulose causes bloating; macrogol is often better tolerated.

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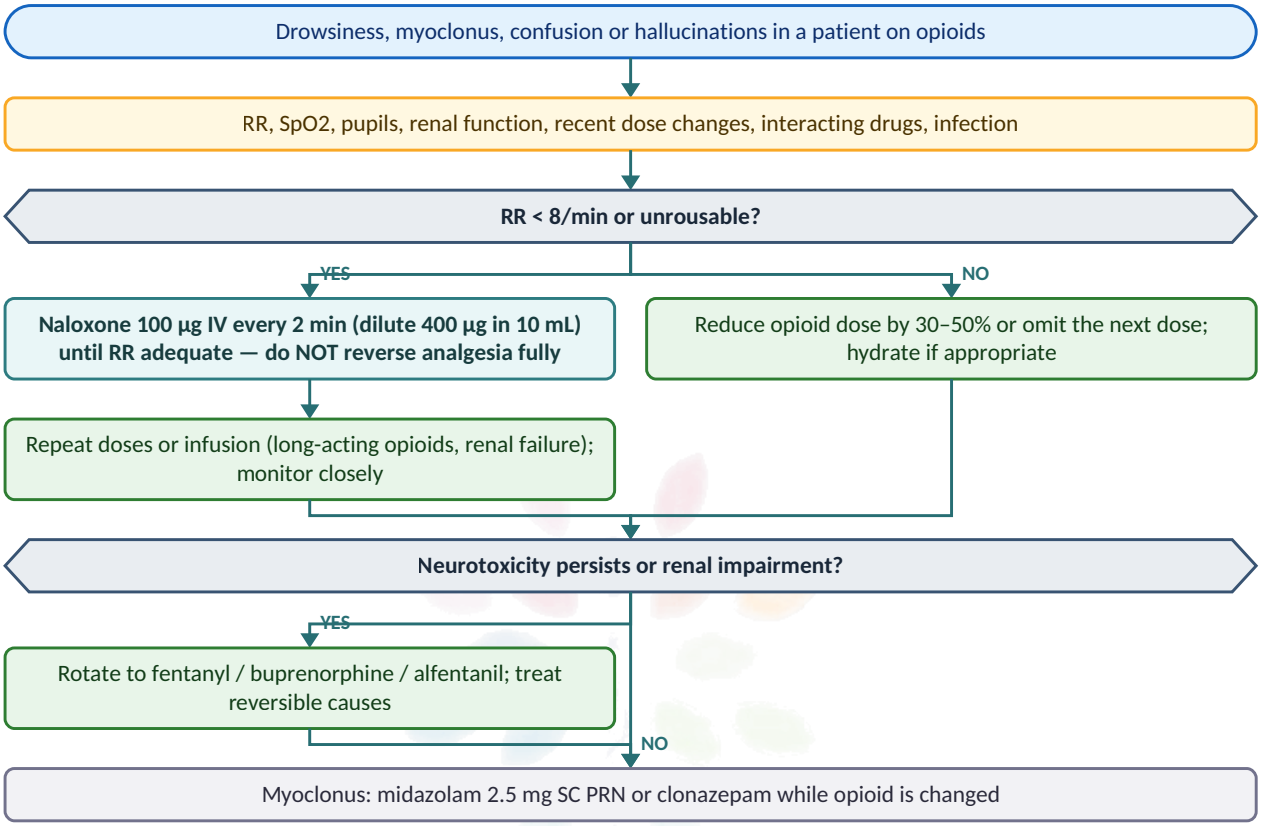
MODEL SHORT-ESSAY ANSWER · SE 26

Opioid-Induced Neurotoxicity

Read the text ›

Opioid-induced neurotoxicity is a syndrome of sedation, confusion/delirium, myoclonus, hyperalgesia, and (rarely) seizures, resulting from accumulation of opioid or active metabolites, particularly in renal impairment, dehydration, or rapid dose escalation. It is distinguished from simple undertreated pain by the combination of neuroexcitatory features alongside sedation, sometimes with paradoxically worsening pain (opioid-induced hyperalgesia). Management includes dose reduction, hydration, opioid rotation to an opioid with fewer active metabolites (e.g. fentanyl in renal impairment), and symptomatic treatment of myoclonus if distressing.

ALGORITHM · Opioid Toxicity and Opioid-Induced Neurotoxicity Algo 24 — full page ›



★ KEY POINTS

- Full-dose naloxone in opioid-tolerant patients causes severe pain and withdrawal.
- Morphine-3- and -6-glucuronide accumulate in renal failure.

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MODEL SHORT-ESSAY ANSWER · SE 27

Methadone — Special Pharmacology

Read the text ›

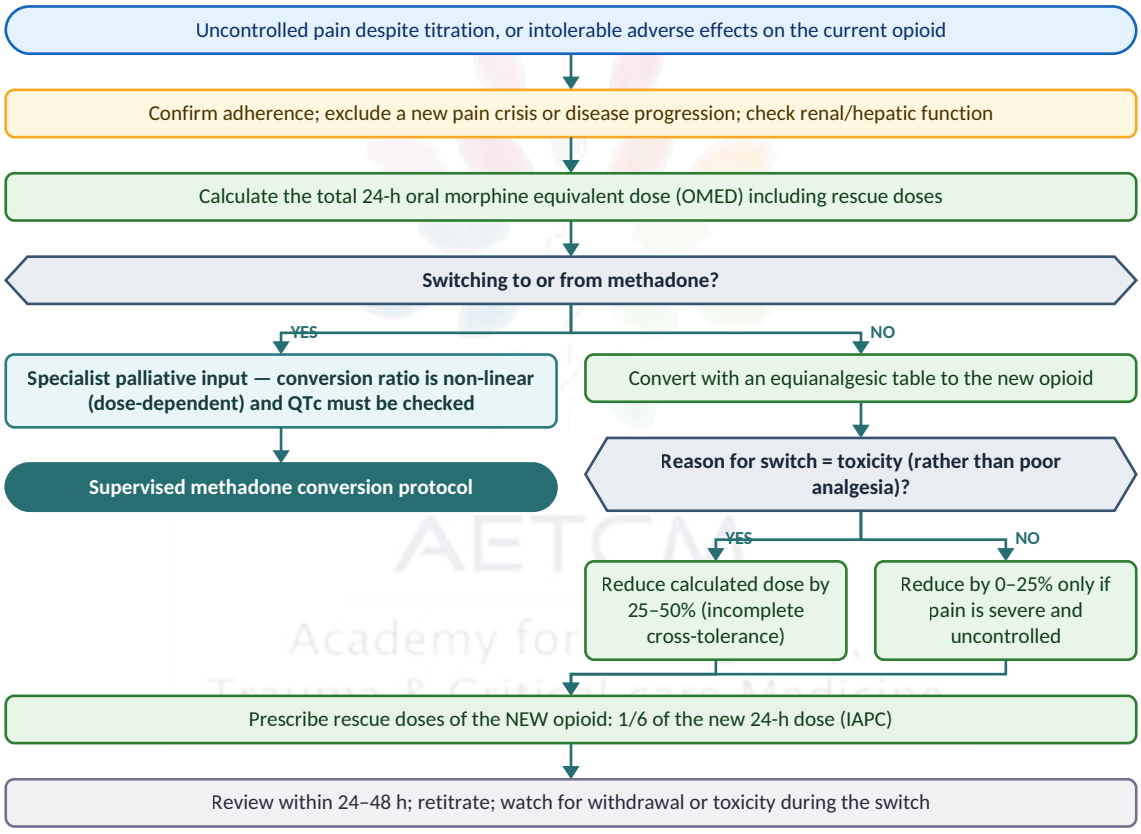
Methadone is a synthetic opioid with mu-agonist activity plus NMDA-receptor antagonism (useful in neuropathic/refractory pain) and serotonin-noradrenaline reuptake inhibition, but its use is complicated by a long and highly variable half-life (8–60 hours, risking delayed accumulation and over-sedation days after starting), a non-linear, dose-dependent equianalgesic ratio to morphine that increases at higher pre-rotation doses, and a risk of QT-interval prolongation. For these reasons methadone initiation and rotation should only be undertaken by or with specialist palliative medicine input, with slow titration and ECG monitoring where risk factors for QT prolongation exist.

EXAM-ORIENTED THEORY

Methadone — key facts

Aspect	Detail
Mechanism	μ-opioid agonist + NMDA antagonist + SNRI activity
Pharmacokinetics	High oral bioavailability (~80%); long, variable half-life (8–60 h); steady state 5–7 days
Conversion	Ratio depends on prior dose (e.g. 4:1 at OMED < 90 mg up to 20:1 at very high doses) — specialist only
Advantages	Neuropathic pain, renal failure, cheap, no active metabolites
Risks	Delayed accumulation, QTc prolongation, CYP3A4 interactions (rifampicin, azoles)
Monitoring	ECG at baseline and after dose changes; review daily during titration

ALGORITHM · Opioid Rotation Algo 2 — full page ›



★ KEY POINTS

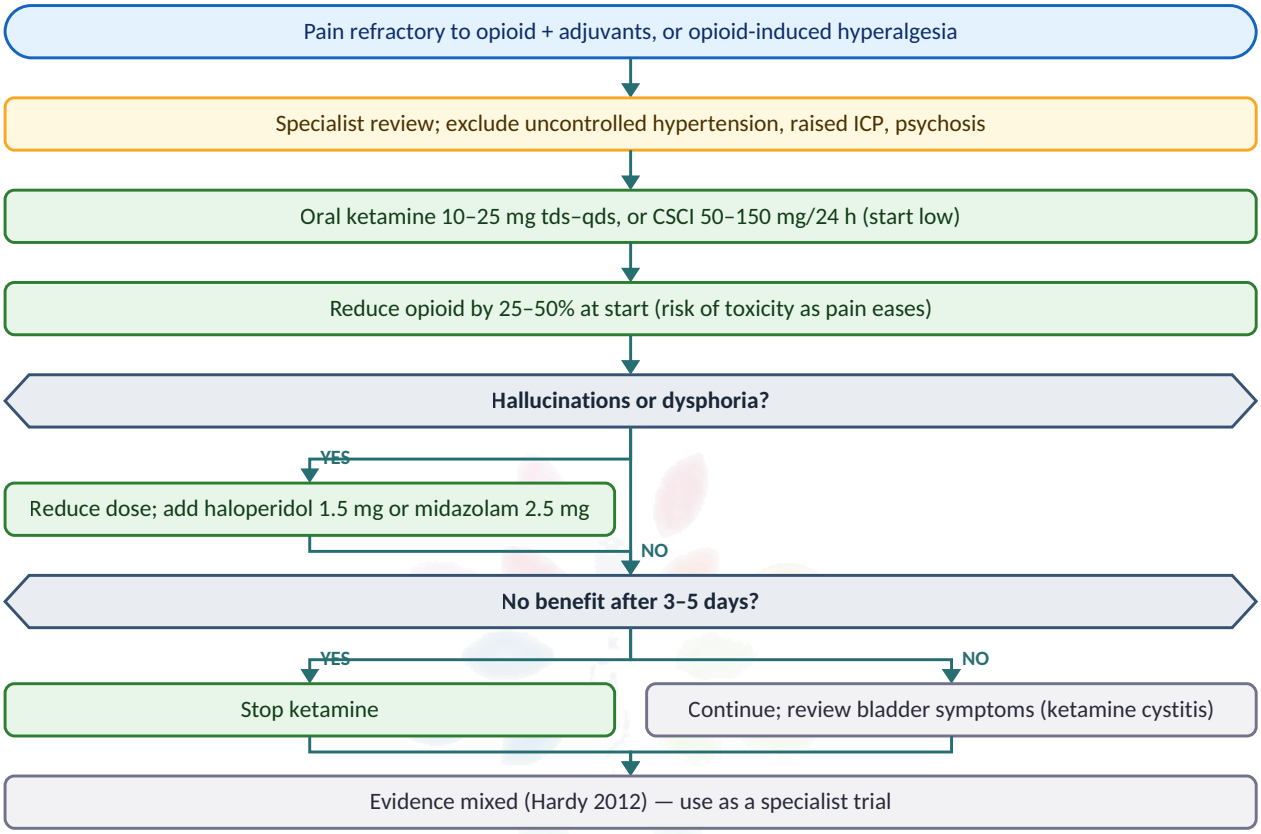
- ► Fentanyl patch: start new patch and stop the old opioid at the appropriate time (12 h overlap for SR morphine).
- ► Renal failure is the commonest reason to rotate from morphine — choose fentanyl, buprenorphine or alfentanil.

MODEL SHORT-ESSAY ANSWER · SE 28

Ketamine in Refractory Pain

Ketamine, an NMDA-receptor antagonist, is used as an adjuvant analgesic for pain refractory to optimised opioid and standard adjuvant therapy, particularly pain with a central sensitisation/neuropathic component (opioid-induced hyperalgesia, refractory neuropathic or incident pain). It can be given orally, subcutaneously, or intravenously, usually at sub-anaesthetic doses, often as a burst or continuous low-dose infusion. Adverse effects include dissociative/psychomimetic symptoms (vivid dreams, hallucinations, dysphoria), hypertension, and sedation, generally requiring specialist-level monitoring; it is reserved as a later-line option rather than routine first-line therapy.

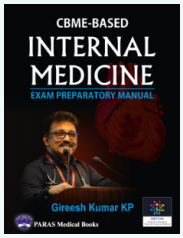
ALGORITHM · Ketamine for Refractory Pain Algo 52 — full page ›



★ KEY POINTS

- Sub-anaesthetic doses act on NMDA-mediated central sensitisation.
- Burst ketamine protocols are used in some centres.

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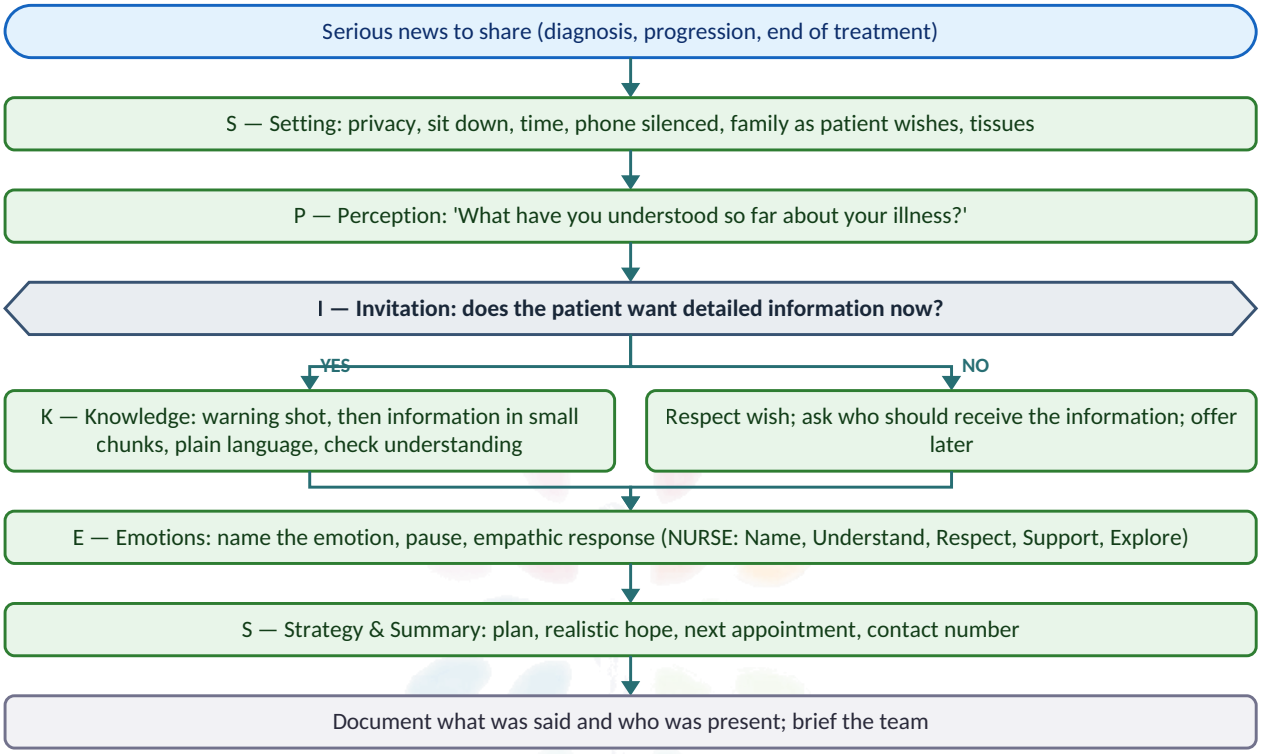
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MODEL SHORT-ESSAY ANSWER · SE 29

Communication — the SPIKES Protocol in Detail

SPIKES structures the delivery of difficult news: **S**etting (privacy, adequate uninterrupted time, appropriate seating and eye contact); **P**erception (ask what the patient already understands); **I**nvitation (establish how much detail the patient wants); **K**nowledge (deliver information in small chunks using plain language, with a brief 'warning shot' before the key news); **E**motions (name and respond empathetically to the patient's emotional reaction before continuing with information); and **S**trategy/Summary (agree next steps and ensure ongoing availability). Used consistently, SPIKES improves patient understanding, satisfaction, and trust, and is a core, examinable communication skill.

ALGORITHM · Breaking Bad News — the SPIKES Protocol Algo 12 — full page ›



★ KEY POINTS

- Silence is a skill — allow it after the news.
- In India, families often request non-disclosure — explore their fears and the patient's wishes, don't simply comply.

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MODEL SHORT-ESSAY ANSWER · SE 30

Palliative Care in Pandemic / Resource-Constrained Crisis Settings

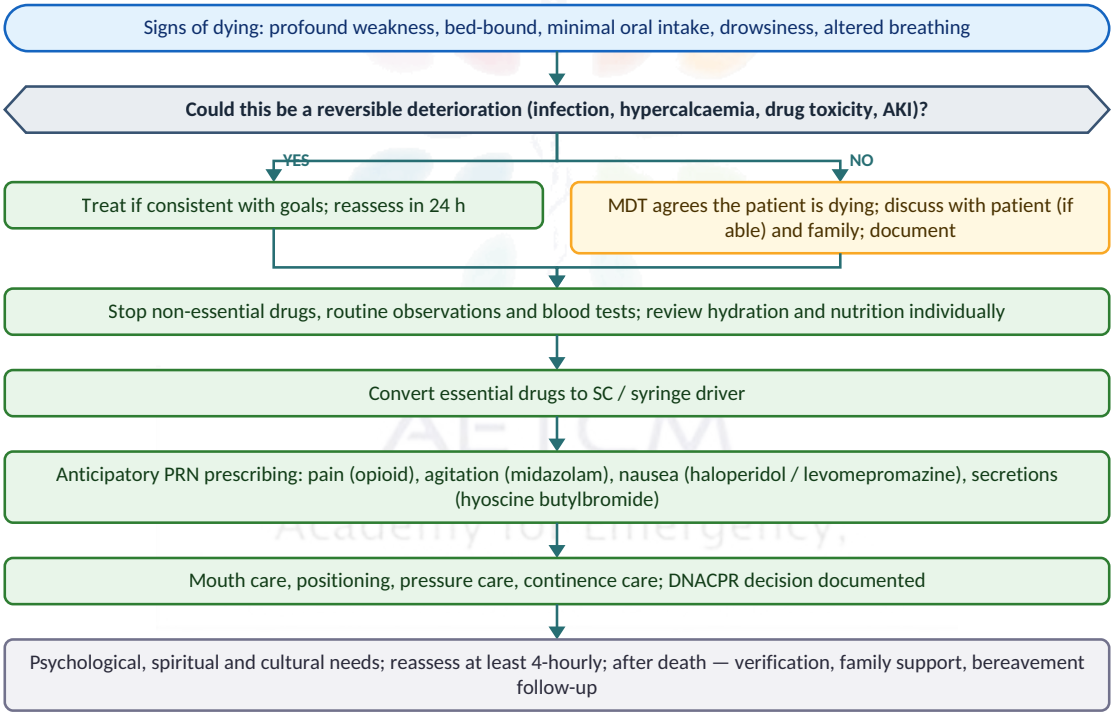
Pandemics and public health emergencies (as seen during COVID-19) place unique strain on palliative care delivery — visitor restrictions separating dying patients from family, overwhelmed services limiting time for communication, rapid symptom escalation requiring efficient protocol-based management, and a surge in bereavement needs. Adaptations include structured remote/video communication to maintain family connection where in-person visits are restricted, clear anticipatory symptom-control protocols for rapidly deteriorating patients, telemedicine-supported home palliative care to reduce hospital burden, and proactive psychological support for both bereaved families and exhausted frontline staff. These experiences have reinforced the importance of early integration of palliative care principles into crisis response planning.

EXAM-ORIENTED THEORY

Palliative care in crises and resource-limited settings

Domain	Essential response
Triage	Integrate palliative care into triage for patients unlikely to benefit from scarce critical care
Essential medicines	Morphine, midazolam, haloperidol, hyoscine, dexamethasone, paracetamol
Communication	Video/phone family meetings; clear, compassionate updates
Staff	Train non-specialists; protect staff wellbeing
Bereavement	Remote rituals; outreach to families unable to visit
Reference	WHO 'Integrating palliative care in humanitarian emergencies' (2018)

ALGORITHM · Care in the Last Hours of Life (The Dying Phase) Algo 7 — full page ›



★ KEY POINTS

- Recognising dying is a team diagnosis — revisit it if the patient improves.
- Rituals (e.g. Ganga jal, prayers) matter to Indian families — ask, don't assume.

VIVA VOCE RAPID-FIRE · Viva 1

CBME — Introduction & Principles of Palliative Medicine

Read the text ›

Q. Define palliative care as per WHO.

An approach that improves quality of life of patients and families facing life-threatening illness, through prevention and relief of suffering by early identification, assessment and treatment of pain and other physical, psychosocial and spiritual problems.

Q. Who is considered the founder of the modern hospice movement?

Dame Cicely Saunders, who founded St Christopher's Hospice in London in 1967.

Q. What is 'total pain'?

Saunders' concept that suffering has physical, psychological, social and spiritual components, all requiring attention.

Q. What distinguishes palliative care from hospice care?

Palliative care can be given alongside curative treatment from diagnosis onward; hospice care is typically reserved for patients with a limited prognosis and focuses solely on comfort.

Q. Name the levels at which palliative care can be delivered in India.

Primary (general physicians/PHCs), secondary/specialist (district/tertiary hospital units), and tertiary/academic (teaching hospital MD/DNB programmes).

Q. What is the Neighbourhood Network in Palliative Care?

Kerala's community-owned, volunteer-driven model of home-based palliative care, widely cited for achieving high population coverage at low cost.

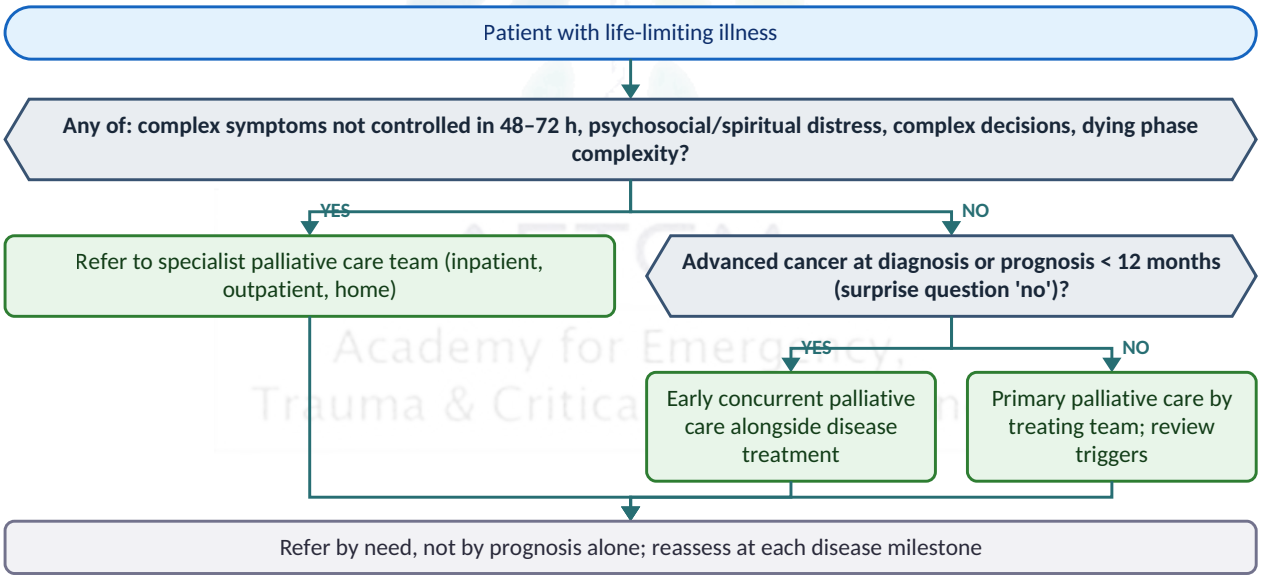
Q. Name the key 2014 legislative change improving opioid access in India.

Amendment to the Narcotic Drugs and Psychotropic Substances (NDPS) Act simplifying licensing for recognised healthcare institutions to stock and dispense morphine.

Q. What are the four domains of 'total pain'?

Physical, psychological, social, and spiritual.

ALGORITHM · When to Refer to Specialist Palliative Care Algo 53 — full page ›



VIVA VOCE RAPID-FIRE · Viva 2

CBME — Ethics, Law and Communication

Q. What did the Common Cause vs Union of India (2018) judgment establish?
Legalised passive euthanasia and permitted execution of an Advance Medical Directive (living will) by competent adults, as part of the right to die with dignity under Article 21.

Q. Is active euthanasia legal in India?
No, active euthanasia and physician-assisted suicide remain illegal in India.

Q. What is the doctrine of double effect?
An ethically permissible action with a good intended effect (symptom relief) that also has a foreseen but unintended harmful effect, provided the harm is not the means to the good effect and the intervention is proportionate.

Q. What are the four principles of biomedical ethics?
Autonomy, beneficence, non-maleficence, and justice.

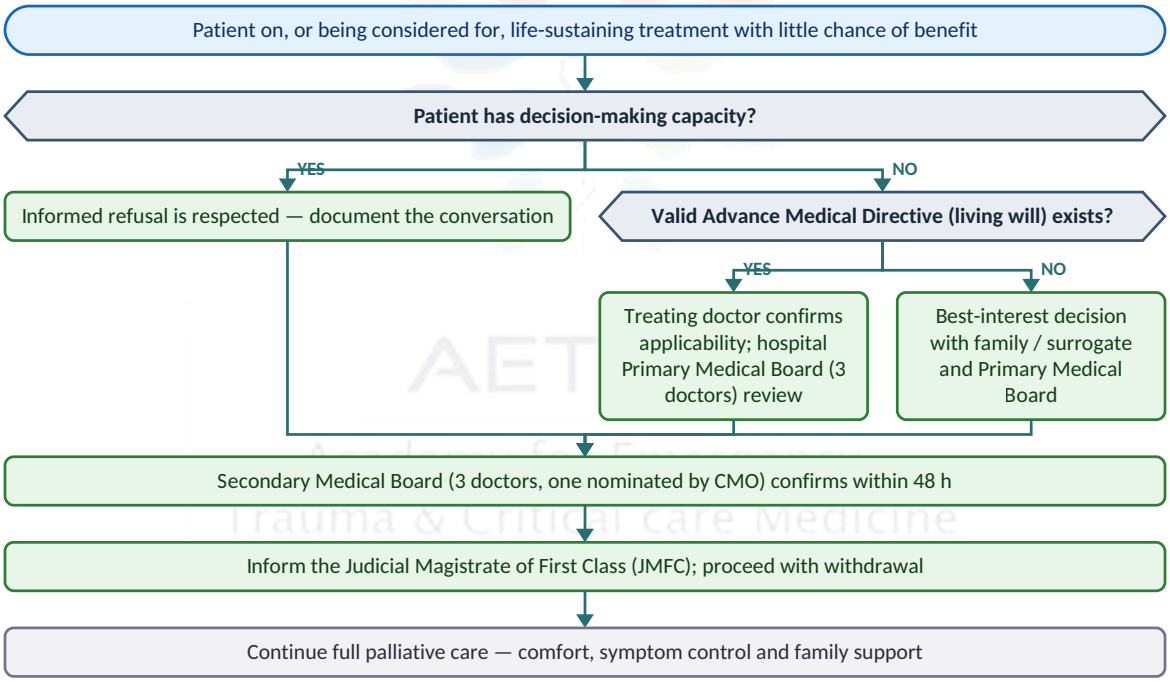
Q. Expand the acronym SPIKES.
Setting, Perception, Invitation, Knowledge, Emotions, Strategy/Summary — the protocol for breaking bad news.

Q. Is withdrawing life-sustaining treatment the same as euthanasia?
No, withdrawing or withholding futile/disproportionately burdensome treatment is ethically and legally distinct from euthanasia, as death results from the underlying disease, not a deliberate act to cause death.

Q. What simplification did the 2023 Supreme Court ruling make?
It simplified the cumbersome multi-layer medical board/judicial approval process from 2018 for executing and implementing an Advance Medical Directive.

Q. Name a tool for spiritual history-taking.
FICA (Faith, Importance, Community, Address in care) or HOPE.

ALGORITHM · Withholding or Withdrawing Life-Sustaining Treatment in India Algo 36 — full page ›



★ KEY POINTS

- Withdrawal of futile treatment is not euthanasia — intent is to stop burdensome treatment, not to cause death.
- The 2023 modification removed the need for magistrate countersignature of living wills (notary/gazetted officer attestation suffices).

VIVA VOCE RAPID-FIRE · Viva 3

CBME — Psychosocial, Nursing and Rehabilitative Care

Q. Name the five Kubler-Ross stages.

Denial, anger, bargaining, depression, acceptance.

Q. What are Worden's four tasks of mourning?

Accepting the reality of the loss, processing the pain of grief, adjusting to a world without the deceased, and finding an enduring connection while moving forward.

Q. What defines complicated/prolonged grief disorder?

Persistent, intense yearning and functional impairment extending well beyond expected cultural/social norms, generally beyond 6–12 months.

Q. What is caregiver burnout?

Exhaustion, depression, and resentment resulting from unaddressed, sustained caregiver burden.

Q. What is compassion fatigue?

Cumulative emotional/physical exhaustion in healthcare providers from sustained empathetic engagement with suffering, distinct from but related to general burnout.

Q. What validated tool assesses pain in advanced dementia?

PAINAD (Pain Assessment in Advanced Dementia).

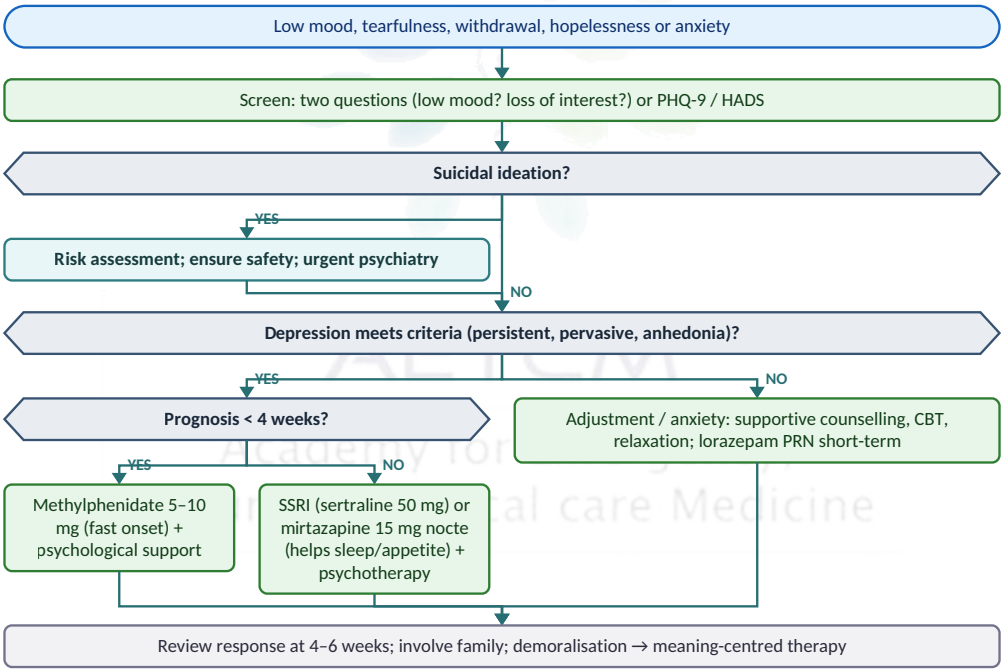
Q. What validated tool assesses pain in young children?

FLACC (Face, Legs, Activity, Cry, Consolability).

Q. Name one role of the medical social worker in a palliative team.

Addressing financial assistance, family dynamics, discharge planning, and practical/legal issues.

ALGORITHM · Depression and Anxiety in Palliative Care Algo 54 — full page ›



★ KEY POINTS

- Somatic symptoms (fatigue, anorexia) are unreliable markers in cancer — focus on cognitive features.
- Mirtazapine is versatile in palliative care.

VIVA VOCE RAPID-FIRE · Viva 4

Pain — Basic Science & Assessment

Read the text ›

Q. What are the four steps of the pain pathway?
Transduction, transmission, modulation, perception.

Q. Differentiate peripheral and central sensitisation.
Peripheral sensitisation occurs at the nociceptor terminal from inflammatory mediators (primary hyperalgesia); central sensitisation occurs in dorsal horn neurons via NMDA-receptor-mediated wind-up (secondary hyperalgesia/allodynia).

Q. Expand SOCRATES.
Site, Onset, Character, Radiation, Associations, Time course, Exacerbating/relieving factors, Severity.

Q. Name three validated unidimensional pain scales.
Numerical Rating Scale (NRS), Visual Analogue Scale (VAS), Wong-Baker FACES scale.

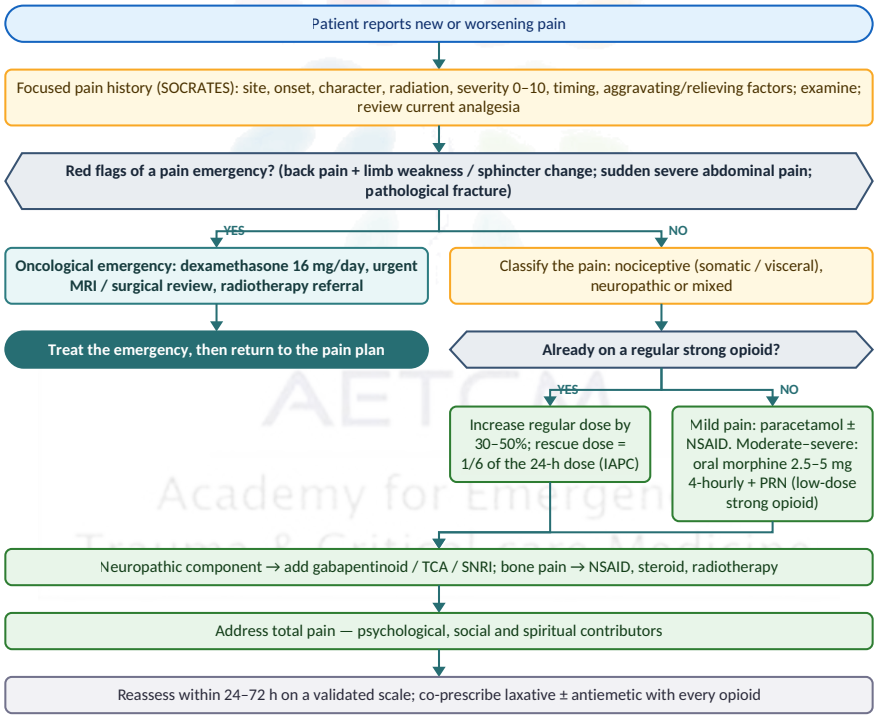
Q. Name a multidimensional pain assessment tool.
Brief Pain Inventory (BPI) or McGill Pain Questionnaire.

Q. What tool is used for critically ill, ventilated patients unable to self-report pain?
Critical-Care Pain Observation Tool (CPOT).

Q. What does ESAS stand for and assess?
Edmonton Symptom Assessment System — a multi-symptom self-report tool covering pain, fatigue, nausea, and other common symptoms.

Q. What is allodynia?
Pain caused by a stimulus that does not normally provoke pain, e.g. light touch.

ALGORITHM · Approach to New or Worsening Pain in a Palliative Care Patient Algo 1 — full page ›



★ KEY POINTS

- Always exclude a pain emergency first — spinal cord compression and pathological fracture are time-critical.
- Titrate by 30–50% steps; 'by the clock, by the mouth, by the ladder, for the individual'.

VIVA VOCE RAPID-FIRE · Viva 5

Pain — Pharmacological Management

Read the text ›

Q. State the WHO analgesic ladder steps.
Step 1: non-opioid ± adjuvant for mild pain. Step 2: weak opioid + Step 1 agents for mild-moderate pain. Step 3: strong opioid + Step 1 agents for moderate-severe pain.

Q. Name the three opioid receptor types and their main effect.
Mu (main analgesic target, also euphoria/respiratory depression/constipation), kappa (spinal analgesia, dysphoria), delta (analgesia, mood modulation).

Q. Which opioids are preferred in renal impairment and why?
Fentanyl and buprenorphine, as they lack clinically significant active renally-excreted metabolites, unlike morphine (M6G/M3G accumulation).

Q. What is the equianalgesic oral morphine : oral tramadol ratio (approximate)?
Approximately 1:10.

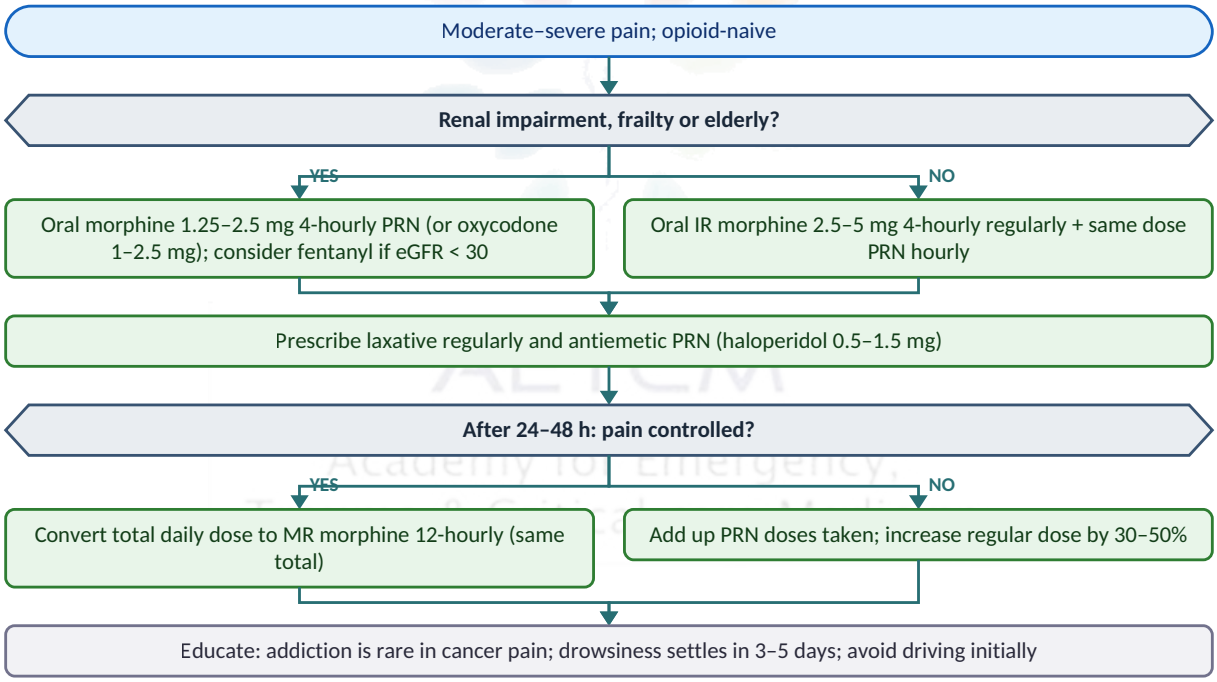
Q. What first-line laxative combination is used prophylactically with opioids?
Stimulant (senna) plus softener (docusate).

Q. What drug reverses opioid-induced respiratory depression?
Naloxone, given in small titrated IV boluses (0.04–0.1 mg) to avoid precipitating acute withdrawal/pain crisis.

Q. Name a peripherally-acting mu-opioid receptor antagonist used for refractory opioid-induced constipation.
Methylnaltrexone.

Q. What distinguishes methadone pharmacologically from other opioids?
It has additional NMDA-receptor antagonist activity, a long and variable half-life, and a non-linear, dose-dependent equianalgesic ratio.

ALGORITHM · Starting Opioids in an Opioid-Naive Patient Algo 29 — full page ›



★ KEY POINTS

- Weak opioids (step 2) can be skipped — low-dose morphine is as effective (Bandieri 2016).
- Oral morphine bioavailability ~30%.

VIVA VOCE RAPID-FIRE · Viva 6

Pain — Interventional and Emergency Management

Read the text ›

Q. Name an indication for coeliac plexus block.
Refractory pancreatic cancer pain.

Q. What agents are used for neurolytic (long-duration) nerve blocks?
Absolute alcohol or phenol.

Q. What is the first step in managing suspected spinal cord compression?
Immediate high-dose dexamethasone, even before imaging, followed by urgent whole-spine MRI.

Q. What determines choice between surgery and radiotherapy for cord compression?
Performance status, single vs multiple compression levels, spinal instability, and tumour radiosensitivity.

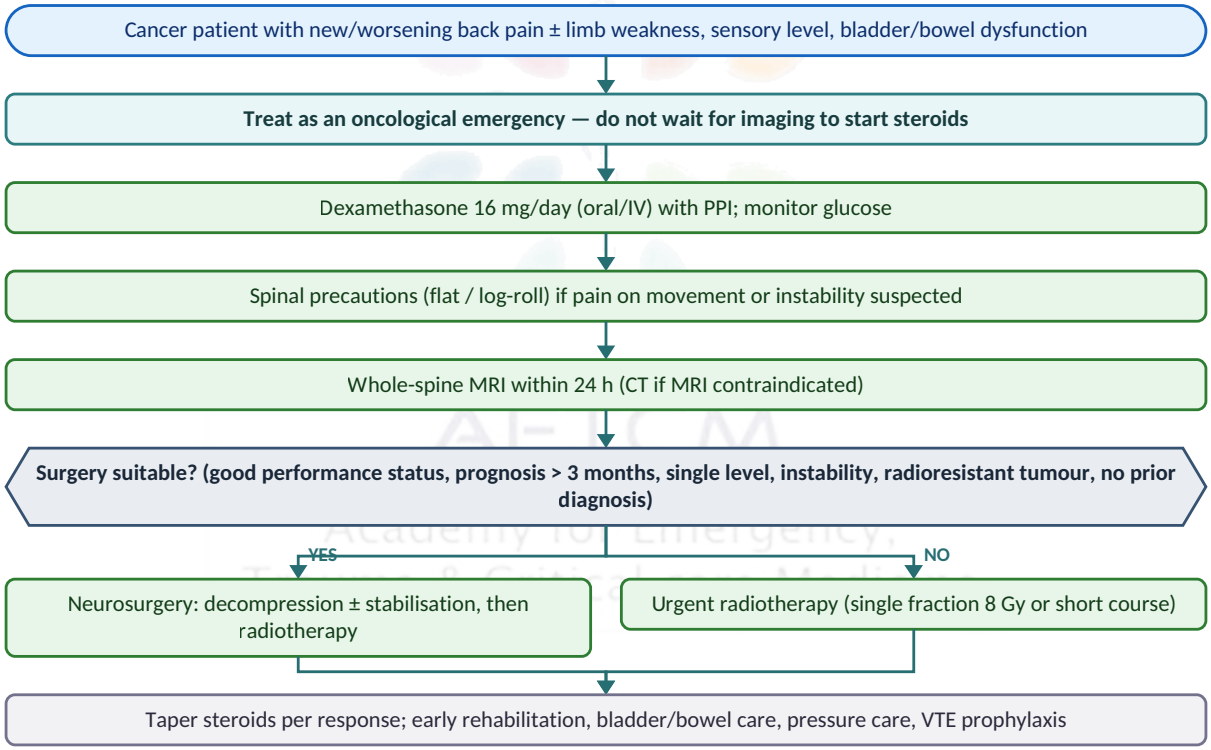
Q. What is the immediate management of severe symptomatic hypercalcaemia of malignancy?
Aggressive IV isotonic saline rehydration, followed by an IV bisphosphonate once rehydrated; calcitonin for rapid short-term effect if needed.

Q. What defines pseudoaddiction?
Drug-seeking behaviour that occurs due to undertreated pain and resolves once pain is adequately controlled, distinguishing it from true opioid use disorder.

Q. Which Indian act regulates access to opioid analgesics?
The Narcotic Drugs and Psychotropic Substances (NDPS) Act, amended in 2014 to simplify institutional licensing.

Q. Name a radiopharmaceutical used for widespread painful bone metastases.
Strontium-89 or Samarium-153.

ALGORITHM · Approach to Suspected Malignant Spinal Cord Compression Algo 9 — full page ›



★ KEY POINTS

- Ambulatory status at diagnosis is the strongest predictor of outcome.
- Patchell trial (2005): surgery + RT kept more patients walking than RT alone in selected cases.

VIVA VOCE RAPID-FIRE · Viva 7

Non-Pain Symptom Management

Q. Name the four receptor pathways relevant to the choice of antiemetic.
Dopamine (D2) and serotonin (5-HT3) for chemical/metabolic causes, histamine (H1) and acetylcholine (ACh) for vestibular causes, 5-HT4/D2 for gastric stasis, ACh/H1 for mechanical obstruction.

Q. First-line drug for terminal respiratory secretions ('death rattle')?
An anticholinergic such as hyoscine butylbromide, hyoscine hydrobromide, or glycopyrrolate, started early (anticipatory prescribing).

Q. First-line drug for terminal delirium/agitation?
Haloperidol.

Q. Why avoid benzodiazepines alone as first-line for delirium?
They can worsen or precipitate delirium, particularly the hypoactive subtype.

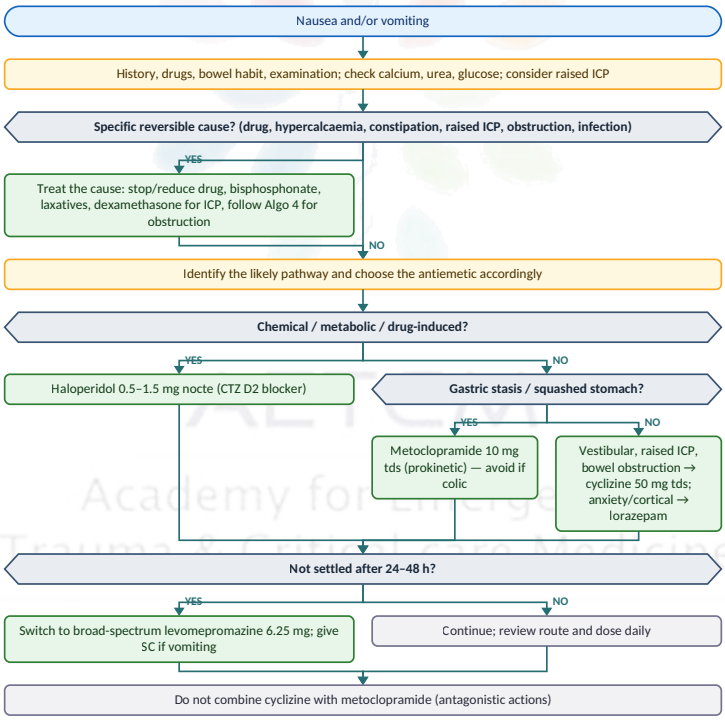
Q. What non-pharmacological measure has evidence for refractory breathlessness?
A hand-held fan directed at the face.

Q. What is the evidence for tube feeding in advanced dementia?
It does not prolong survival, prevent aspiration pneumonia, or improve quality of life compared with careful hand feeding.

Q. Name a drug used for cancer-related fatigue in selected refractory cases.
Methylphenidate (a psychostimulant), with modest evidence.

Q. What is the treatment of choice for anaerobic-related malodour in fungating wounds?
Topical or systemic metronidazole.

ALGORITHM · Approach to Nausea and Vomiting in Palliative Care Algo 8 — full page ›



★ KEY POINTS

- Choose antiemetic by mechanism, not by habit.
- Ondansetron is mainly for chemo-/radiotherapy-induced emesis and constipates.

VIVA VOCE RAPID-FIRE · Viva 8

Pediatric and Non-Malignant Palliative Care

Q. What is the WHO two-step approach for pediatric pain?
Non-opioids for mild pain, moving directly to strong opioids for moderate-severe pain, skipping weak opioids.

Q. Name a validated pain scale for infants/non-verbal children.
FLACC or CRIES.

Q. How does the disease trajectory of organ failure differ from cancer?
Organ failure shows gradual decline with acute exacerbations and unpredictable death during a crisis, versus cancer's relatively predictable late rapid decline.

Q. What should be discussed near end of life in a patient with an implantable defibrillator?
Deactivation of the defibrillator to prevent distressing shocks during the dying process.

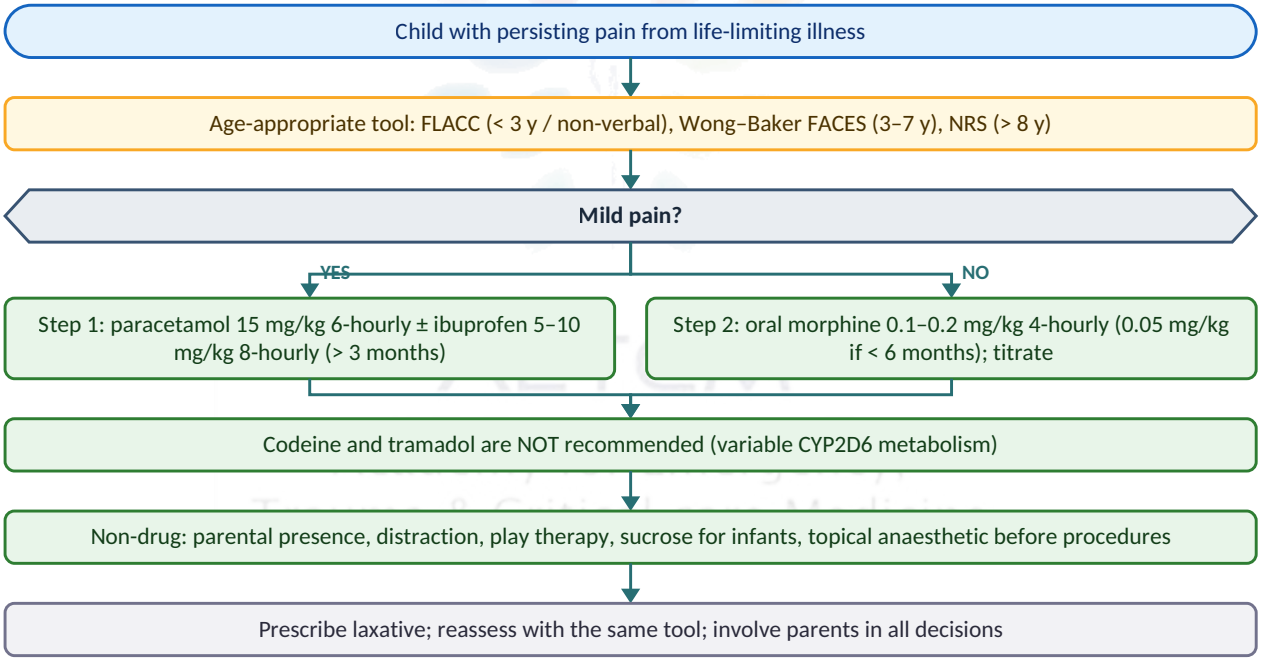
Q. What palliative pathway is relevant to patients choosing not to pursue dialysis in ESRD?
Conservative (non-dialysis) kidney management with integrated palliative symptom control and advance care planning.

Q. Why is prognostication harder in non-malignant disease?
Trajectories are less predictable, with death often occurring unexpectedly during an acute exacerbation rather than a terminal decline phase.

Q. What opioid considerations apply to ESRD patients?
Prefer opioids without active renally-excreted metabolites (fentanyl, buprenorphine), start at low doses, titrate cautiously.

Q. Who should be the unit of care in pediatric palliative care?
The whole family, including siblings, not the child patient alone.

ALGORITHM · Pain Management in Children Algo 32 — full page ›



★ KEY POINTS

- Children are not small adults — dose by weight and age.
- ACT categories 1–4 classify life-limiting conditions for paediatric palliative care.

VIVA VOCE RAPID-FIRE · Viva 9

Non-Pharmacological and Rehabilitative Approaches

Read the text ›

Q. Name three non-pharmacological modalities for pain.
TENS (transcutaneous electrical nerve stimulation), physiotherapy, complete decongestive therapy (for lymphedema-related discomfort).

Q. What is the purpose of cognitive-behavioural therapy in pain management?
Restructuring catastrophising thoughts about pain and building coping/self-efficacy.

Q. Name a psychological therapy shown to help procedural/anticipatory pain and anxiety.
Music therapy.

Q. What does complete decongestive therapy for lymphedema include?
Manual lymphatic drainage, multilayer compression bandaging, maintenance compression garments, skin care, and remedial exercise.

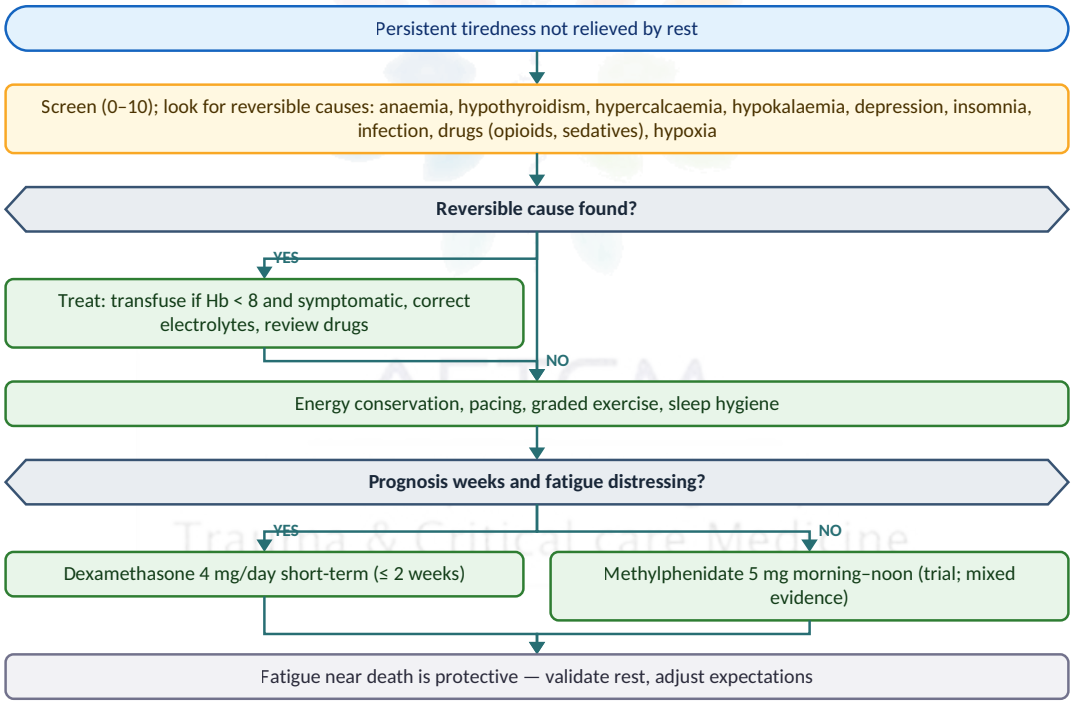
Q. Are diuretics effective for lymphedema?
No, lymphedema fluid is protein-rich and diuretics are generally ineffective; not routinely recommended.

Q. What four domains should a holistic pain answer explicitly link to?
Physical, psychological, social, and spiritual (the domains of total pain).

Q. Name an integrative therapy sometimes used as a disclosed adjunct in palliative pain management.
Yoga or acupuncture/acupressure, always disclosed to and coordinated with the primary treating team.

Q. What is guided imagery used for?
Reducing anxiety and pain perception through structured relaxation/visualisation techniques.

ALGORITHM · Fatigue in Palliative Care Algo 49 — full page ›



★ KEY POINTS

- Exercise has the strongest evidence for cancer-related fatigue in earlier disease.
- Steroid benefit wears off after 2–4 weeks.

VIVA VOCE RAPID-FIRE · Viva 10

Assessment, Prognostication and Models of Care

Q. Name two composite prognostic tools used in advanced cancer.
Palliative Prognostic Index (PPI) and Palliative Prognostic Score (PaP score).

Q. What functional scale correlates performance status with survival?
Palliative Performance Scale or Karnofsky Performance Status.

Q. What clinical signs suggest a prognosis of days (the dying phase)?
Profound weakness, minimal oral intake, altered consciousness, and changed breathing pattern.

Q. What is anticipatory prescribing?
Pre-emptively prescribing medications (for pain, agitation, nausea, secretions) likely to be needed in the dying phase, so they are available without delay when symptoms arise.

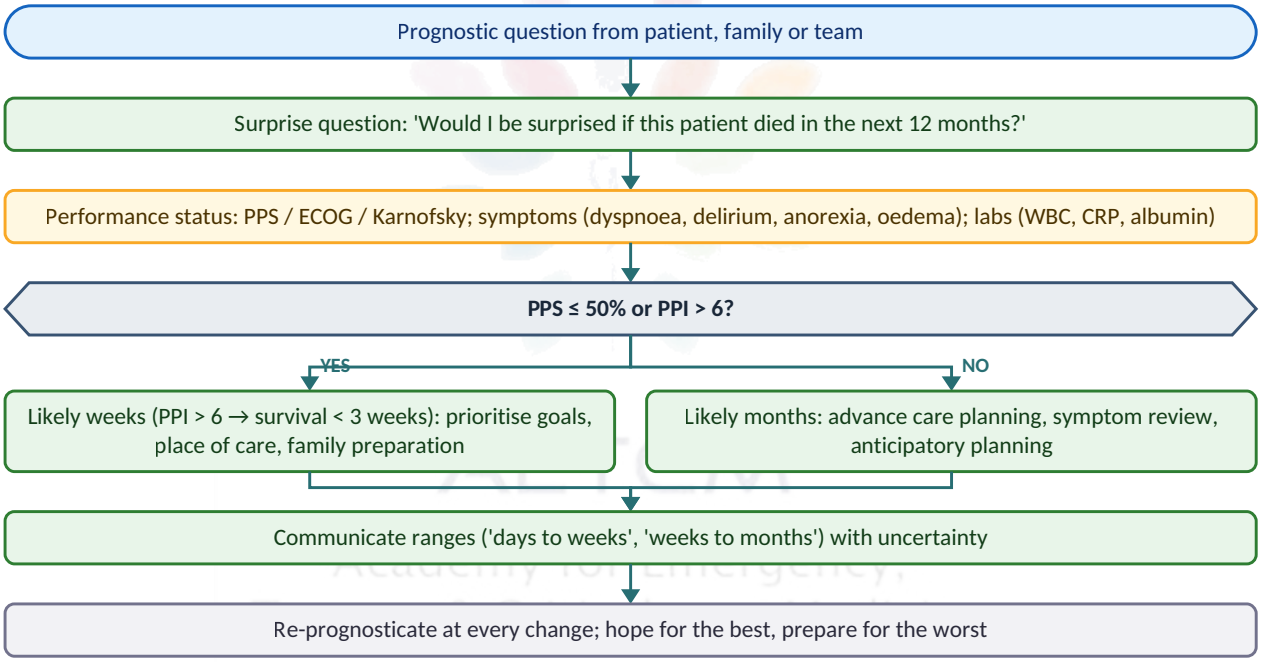
Q. What route is preferred for medication administration in the dying phase when oral intake fails?
Subcutaneous, often via continuous infusion (syringe driver).

Q. What four drug classes are commonly combined in a syringe driver?
Opioid (analgesia), antiemetic, anticholinergic (secretions), and benzodiazepine (agitation).

Q. What is the key message to give families about artificial nutrition/hydration in the actively dying patient?
It confers no proven benefit and may increase discomfort/secretions; comfort-focused care is preferred.

Q. Name one barrier to palliative care integration in non-malignant disease.
Prognostic uncertainty and under-recognition of the life-limiting nature of the condition by treating specialists.

ALGORITHM · Prognostication in Advanced Illness Algo 48 — full page ›



★ KEY POINTS

- PaP score uses dyspnoea, anorexia, KPS, CPS, WBC and lymphocyte %.
- Rate of change in function is a powerful prognostic clue.

MCQ QUESTION BANK · MCQ 1

Principles of Palliative Care & Ethics

1. Who founded the modern hospice movement and St Christopher's Hospice?

A. Elisabeth Kubler-Ross
B. Cicely Saunders
C. Florence Wald
D. Balfour Mount

Answer: B. Cicely Saunders

Dame Cicely Saunders founded St Christopher's Hospice (1967) and introduced the concept of total pain.

2. Which Supreme Court judgment legalised passive euthanasia and the Advance Medical Directive in India?

A. Aruna Shanbaug (2011)
B. Common Cause vs Union of India (2018)
C. K.S. Puttaswamy (2017)
D. Gian Kaur (1996)

Answer: B. Common Cause vs Union of India (2018)

Common Cause vs Union of India (2018) legalised passive euthanasia and permitted execution of a living will, later simplified in 2023.

3. Active euthanasia and physician-assisted suicide in India are:

A. Fully legal with consent
B. Legal only in terminal illness
C. Illegal
D. Legal only with court approval each time

Answer: C. Illegal

Active euthanasia and physician-assisted suicide remain illegal in India; only passive withdrawal/withholding is permitted.

4. The doctrine of double effect ethically justifies:

A. Active euthanasia
B. Appropriately titrated opioid/sedative use for symptom relief despite a theoretical risk of hastening death
C. Physician-assisted suicide
D. Withholding information from a competent patient

Answer: B. Appropriately titrated opioid/sedative use for symptom relief despite a theoretical risk of hastening death

The doctrine justifies proportionate symptom-relieving treatment where the intent is relief of suffering, not death, even with a foreseen but unintended risk.

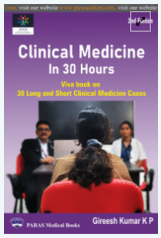
5. Which is NOT one of the four principles of biomedical ethics?

A. Autonomy
B. Beneficence
C. Confidentiality
D. Justice

Answer: C. Confidentiality

The four classical principles are autonomy, beneficence, non-maleficence, and justice; confidentiality is a related but separate professional duty.

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MCQ QUESTION BANK · MCQ 2

Communication and Psychosocial Care

1. In the SPIKES protocol, 'I' stands for:

A. Information
B. Invitation
C. Intervention
D. Impact

Answer: B. Invitation

Invitation refers to establishing how much information the patient wants to know.

2. Which model describes oscillation between loss-oriented and restoration-oriented coping in grief?

A. Kubler-Ross stage model
B. Worden's task model
C. Dual-process model
D. SPIKES model

Answer: C. Dual-process model

The dual-process model (Stroebe and Schut) describes healthy grieving as oscillating between these two coping orientations.

3. Complicated/prolonged grief disorder is generally considered when functional impairment and intense yearning persist beyond:

A. 2 weeks
B. 1 month
C. 6–12 months
D. 5 years

Answer: C. 6–12 months

Persistent, intense impairment beyond roughly 6–12 months, exceeding cultural/social norms, suggests complicated grief requiring referral.

4. FICA is a tool used to assess:

A. Pain in non-verbal patients
B. Spiritual needs
C. Caregiver burden
D. Delirium severity

Answer: B. Spiritual needs

FICA (Faith, Importance, Community, Address in care) is a structured spiritual history tool.

5. Compassion fatigue in palliative care providers is best described as:

A. A sign of poor clinical skill
B. Cumulative emotional/physical exhaustion from sustained empathetic engagement with suffering
C. A normal, inconsequential part of the job needing no intervention
D. Unique to nursing staff only

Answer: B. Cumulative emotional/physical exhaustion from sustained empathetic engagement with suffering

Compassion fatigue is a recognised occupational phenomenon requiring active prevention and support strategies across the whole team.

MCQ QUESTION BANK · MCQ 3

Pain — Basic Science and Assessment

Read the text ›

1. The four steps of the pain pathway, in order, are:

A. Perception, modulation, transmission, transduction
B. Transduction, transmission, modulation, perception
C. Transmission, transduction, perception, modulation
D. Modulation, transduction, transmission, perception

Answer: B. Transduction, transmission, modulation, perception

Transduction (peripheral), transmission (to CNS), modulation (dorsal horn/descending pathways), perception (cortex).

2. NMDA-receptor-mediated 'wind-up' underlies:

A. Peripheral sensitisation
B. Central sensitisation
C. Opioid tolerance only
D. Placebo analgesia

Answer: B. Central sensitisation

Central sensitisation in dorsal horn neurons, mediated by NMDA receptors, underlies secondary hyperalgesia and allodynia.

3. PAINAD is validated for pain assessment in:

A. Infants
B. Advanced dementia
C. Critically ill ventilated adults
D. Adolescents

Answer: B. Advanced dementia

PAINAD (Pain Assessment in Advanced Dementia) uses behavioural observation for non-verbal dementia patients.

4. Which tool assesses pain in critically ill, ventilated adults unable to self-report?

A. FLACC
B. CPOT
C. Wong-Baker FACES
D. ESAS

Answer: B. CPOT

The Critical-Care Pain Observation Tool (CPOT) is validated for this population.

5. SOCRATES is a mnemonic for:

A. Opioid side effects
B. Pain history-taking
C. Breaking bad news steps
D. Prognostic indicators

Answer: B. Pain history-taking

Site, Onset, Character, Radiation, Associations, Time course, Exacerbating/relieving, Severity.

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MCQ QUESTION BANK · MCQ 4

WHO Analgesic Ladder and Opioid Pharmacology

Read the text ›

1. Step 1 of the WHO analgesic ladder for mild pain consists of:

A. Weak opioid alone
B. Strong opioid alone
C. Non-opioid ± adjuvant
D. Neurolytic block

Answer: C. Non-opioid ± adjuvant

Step 1 = non-opioid analgesic (paracetamol/NSAID) with or without an adjuvant.

2. A 58-year-old man with metastatic lung cancer and stage 4 CKD on oral morphine develops sedation and myoclonus. The preferred strong opioid alternative is:

A. Same dose of morphine
B. Fentanyl or buprenorphine
C. Pethidine
D. Codeine

Answer: B. Fentanyl or buprenorphine

Fentanyl and buprenorphine lack clinically significant active renally-excreted metabolites, unlike morphine.

3. Which opioid has NMDA-receptor antagonist activity in addition to mu-agonism?

A. Morphine
B. Fentanyl
C. Methadone
D. Codeine

Answer: C. Methadone

Methadone has additional NMDA-antagonist activity, useful in refractory/neuropathic pain, but complicates its pharmacokinetics.

4. The approximate oral morphine to oral tramadol equianalgesic ratio is:

A. 1:1
B. 1:10
C. 1:100
D. 10:1

Answer: B. 1:10

Approximately 1:10 (always individualised).

5. When rotating opioids, the calculated equianalgesic dose of the new opioid is typically reduced by:

A. No reduction needed
B. 10%
C. 25–50%
D. 90%

Answer: C. 25–50%

A 25–50% reduction accounts for incomplete cross-tolerance between different opioids.

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MCQ QUESTION BANK · MCQ 5

Opioid Adverse Effects and Toxicity

Read the text ›

1. Tolerance develops to most opioid adverse effects EXCEPT:

A. Sedation
B. Nausea
C. Constipation
D. Pruritus

Answer: C. Constipation

Tolerance to constipation does not develop; prophylactic laxatives are required for the duration of opioid therapy.

2. First-line prophylactic laxative regimen for opioid-induced constipation is:

A. Bulk-forming agent alone
B. Stimulant + softener (e.g. senna + docusate)
C. Osmotic agent only, started after constipation develops
D. No prophylaxis needed

Answer: B. Stimulant + softener (e.g. senna + docusate)

A stimulant plus softener, started at the time of opioid initiation, is standard prophylaxis.

3. Naloxone should be given for opioid-induced respiratory depression:

A. As a single large bolus always
B. In small titrated boluses to avoid precipitating severe pain/withdrawal
C. Only via the oral route
D. Only after 24 hours of observation

Answer: B. In small titrated boluses to avoid precipitating severe pain/withdrawal

Small titrated IV boluses (0.04–0.1 mg) aim to restore ventilation while minimising acute withdrawal/pain crisis.

4. A peripherally-acting mu-opioid receptor antagonist used for refractory opioid-induced constipation is:

A. Naloxone
B. Methylnaltrexone
C. Naltrexone
D. Flumazenil

Answer: B. Methylnaltrexone

Methylnaltrexone acts selectively on peripheral gut receptors without reversing central analgesia.

5. Opioid-induced neurotoxicity classically presents with sedation, confusion, and:

A. Bradycardia
B. Myoclonus
C. Hypertension
D. Polyuria

Answer: B. Myoclonus

Myoclonus, along with hyperalgesia and sedation/confusion, characterises opioid neurotoxicity, often from metabolite accumulation.

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MCQ QUESTION BANK · MCQ 6

Adjuvant Analgesics and Neuropathic Pain

Read the text ›

1. First-line pharmacotherapy for neuropathic pain includes:

A. NSAIDs alone
B. Gabapentinoids or tricyclic antidepressants
C. Strong opioids alone
D. Bisphosphonates

Answer: B. Gabapentinoids or tricyclic antidepressants

Gabapentin/pregabalin and amitriptyline/duloxetine are first-line for neuropathic pain.

2. Corticosteroids are particularly useful as an adjuvant analgesic for:

A. Mild osteoarthritis pain
B. Pain from nerve/cord compression or raised intracranial pressure
C. Opioid-induced constipation
D. Chronic lower back pain alone

Answer: B. Pain from nerve/cord compression or raised intracranial pressure

Corticosteroids reduce peritumoural oedema, relieving pain from compression and raised ICP.

3. Ketamine's mechanism of analgesic action is via:

A. Mu-opioid receptor agonism
B. NMDA-receptor antagonism
C. GABA receptor agonism
D. Prostaglandin inhibition

Answer: B. NMDA-receptor antagonism

Ketamine is an NMDA-receptor antagonist, useful in refractory/central-sensitisation pain.

4. A known risk of both bisphosphonates and denosumab is:

A. Hypercalcaemia
B. Osteonecrosis of the jaw
C. Hepatotoxicity
D. QT prolongation

Answer: B. Osteonecrosis of the jaw

Both classes carry a risk of osteonecrosis of the jaw; dental assessment is recommended before starting.

5. Which adjuvant is an NMDA-receptor antagonist also used in refractory neuropathic/incident pain?

A. Amitriptyline
B. Gabapentin
C. Ketamine
D. Dexamethasone

Answer: C. Ketamine

Ketamine, an NMDA antagonist, is reserved for refractory cases given its adverse-effect profile.

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MCQ QUESTION BANK · MCQ 7

Interventional Techniques and Radiotherapy

Read the text ›

1. Coeliac plexus block is most useful for pain from:

A. Bone metastases
B. Pancreatic cancer
C. Peripheral neuropathy
D. Bladder cancer

Answer: B. Pancreatic cancer

Coeliac plexus block targets visceral pain from upper abdominal malignancies, especially pancreatic cancer.

2. Agents used for neurolytic (prolonged) nerve blocks include:

A. Lidocaine only
B. Absolute alcohol or phenol
C. Paracetamol
D. Morphine

Answer: B. Absolute alcohol or phenol

Alcohol and phenol produce longer-duration neurolysis compared with local anaesthetic alone.

3. For uncomplicated painful bone metastases, a single 8 Gy fraction of radiotherapy provides:

A. No benefit compared with placebo
B. Pain relief equivalent to multi-fraction regimens, with resource efficiency
C. Relief only after 3 months
D. Relief only in breast cancer

Answer: B. Pain relief equivalent to multi-fraction regimens, with resource efficiency

A single 8 Gy fraction gives equivalent pain relief to longer courses for uncomplicated bone metastases.

4. A radiopharmaceutical used for widespread painful bone metastases is:

A. Strontium-89
B. Gadolinium
C. Technetium-99m pertechnetate
D. Iodine-131

Answer: A. Strontium-89

Strontium-89 and Samarium-153 are radiopharmaceuticals used for systemic bone pain relief.

5. Neuraxial (epidural/intrathecal) analgesia allows:

A. Higher systemic opioid doses than oral route
B. Delivery of opioid at much lower doses close to the spinal cord
C. Use only in terminal sedation
D. Avoidance of all side effects

Answer: B. Delivery of opioid at much lower doses close to the spinal cord

Neuraxial delivery achieves analgesia at much lower systemic opioid exposure than oral/parenteral routes.

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MCQ QUESTION BANK · MCQ 8

Oncologic Emergencies

Read the text ›

1. The most urgent initial step in suspected malignant spinal cord compression is:

A. Await MRI before any treatment
B. Immediate high-dose corticosteroids
C. Immediate surgery regardless of findings
D. Physiotherapy

Answer: B. Immediate high-dose corticosteroids

High-dose corticosteroids are started immediately, with urgent MRI to follow, without delaying treatment.

2. Immediate management of severe symptomatic hypercalcaemia of malignancy is:

A. IV furosemide alone
B. Aggressive IV isotonic saline rehydration, then IV bisphosphonate
C. Oral calcium restriction only
D. Immediate dialysis in all cases

Answer: B. Aggressive IV isotonic saline rehydration, then IV bisphosphonate

Rehydration first corrects volume depletion and improves renal calcium clearance; bisphosphonate gives sustained effect.

3. Calcitonin's role in hypercalcaemia of malignancy is:

A. Definitive long-term treatment
B. Rapid but short-lived calcium lowering as a bridge
C. First-line in all cases
D. Contraindicated in malignancy

Answer: B. Rapid but short-lived calcium lowering as a bridge

Calcitonin acts rapidly but its effect is short-lived, useful while awaiting bisphosphonate onset.

4. Superior vena cava obstruction classically presents with:

A. Lower limb oedema only
B. Facial/neck swelling and distended neck veins
C. Isolated back pain
D. Haematuria

Answer: B. Facial/neck swelling and distended neck veins

SVC obstruction causes facial/neck swelling, distended neck veins, and breathlessness from impaired venous return.

5. In massive catastrophic tumour-related haemorrhage with no option for intervention, the compassionate response is:

A. Aggressive resuscitation attempts regardless of prognosis
B. Rapid sedation (e.g. midazolam) and use of dark towels, with family support
C. Immediate discharge
D. Withholding all symptom relief

Answer: B. Rapid sedation (e.g. midazolam) and use of dark towels, with family support

Pre-emptive planning with rapid sedation and dark towels (to reduce visual distress) is a recognised compassionate approach.

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MCQ QUESTION BANK · MCQ 9

Non-Pain Symptom Management

1. First-line antiemetic for chemical/metabolic causes of nausea (e.g. drug- or hypercalcaemia-induced) is:

A. Cyclizine
B. Haloperidol
C. Ondansetron only
D. Domperidone only

Answer: B. Haloperidol

Haloperidol (D2 antagonist) targets the chemoreceptor trigger zone implicated in chemical/metabolic nausea.

2. First-line drug for terminal respiratory secretions ('death rattle') is:

A. Haloperidol
B. An anticholinergic (e.g. hyoscine butylbromide)
C. Furosemide
D. Morphine

Answer: B. An anticholinergic (e.g. hyoscine butylbromide)

Anticholinergics reduce secretion production and are more effective started early (anticipatory prescribing).

3. First-line drug for distressing terminal delirium/agitation is:

A. Lorazepam alone
B. Haloperidol
C. Diazepam alone
D. Paracetamol

Answer: B. Haloperidol

Haloperidol is first-line; benzodiazepines alone can worsen delirium, particularly the hypoactive subtype.

4. Evidence-based non-pharmacological measure for refractory breathlessness is:

A. Strict bedrest with no positioning changes
B. A hand-held fan directed at the face
C. High-flow oxygen in all patients regardless of saturation
D. Heavy sedation as first-line

Answer: B. A hand-held fan directed at the face

A fan directed at the face reduces breathlessness via trigeminal afferent stimulation, independent of oxygen saturation.

5. Evidence regarding tube feeding in advanced dementia shows it:

A. Prolongs survival significantly
B. Prevents aspiration pneumonia reliably
C. Does not improve survival, aspiration risk, or quality of life compared with careful hand feeding
D. Is always preferred over hand feeding

Answer: C. Does not improve survival, aspiration risk, or quality of life compared with careful hand feeding

Multiple studies show no survival or aspiration-prevention benefit of tube feeding over careful hand feeding in advanced dementia.

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MCQ QUESTION BANK · MCQ 10

Non-Malignant and Pediatric Palliative Care

1. Compared with cancer, the disease trajectory of chronic organ failure (e.g. heart failure, COPD) is characterised by:

A. A predictable late rapid decline
B. Gradual decline punctuated by unpredictable acute exacerbations, with death often occurring during a crisis
C. No decline until sudden death with no preceding symptoms
D. Identical trajectory to cancer

Answer: B. Gradual decline punctuated by unpredictable acute exacerbations, with death often occurring during a crisis

Organ failure trajectories are far less predictable than cancer, complicating prognostication and referral timing.

2. Near the end of life, discussion about deactivating a device is most relevant in which condition?

A. COPD
B. Implantable cardioverter-defibrillator in heart failure
C. ESRD on conservative management
D. Advanced dementia

Answer: B. Implantable cardioverter-defibrillator in heart failure

Defibrillator deactivation prevents distressing shocks during the natural dying process.

3. The WHO's pediatric analgesic approach for moderate-severe pain recommends:

A. Starting with a weak opioid, as in adults
B. Moving directly from non-opioids to strong opioids (two-step approach)
C. Avoiding all opioids in children
D. Using only topical analgesics

Answer: B. Moving directly from non-opioids to strong opioids (two-step approach)

The WHO two-step approach for children skips weak opioids, moving directly to strong opioids for moderate-severe pain.

4. In pediatric palliative care, the unit of care includes:

A. The child patient only
B. The child and parents only
C. The whole family including siblings
D. Only the primary caregiver

Answer: C. The whole family including siblings

The whole family, including often-overlooked siblings, is considered the unit of care.

5. Conservative (non-dialysis) management of ESRD is an appropriate indication for:

A. Withholding all palliative input
B. Integrated palliative care alongside symptom management and advance care planning
C. Immediate hospice admission only
D. Active euthanasia

Answer: B. Integrated palliative care alongside symptom management and advance care planning

Conservative ESRD management is a recognised indication for integrated, non-malignant palliative care.

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MCQ QUESTION BANK · MCQ 11

Prognostication, Service Models and Anticipatory Care

1. The Palliative Prognostic Index and Palliative Prognostic Score are used to:

A. Diagnose cancer stage
B. Estimate survival prognosis in advanced illness
C. Assess pain intensity
D. Screen for depression

Answer: B. Estimate survival prognosis in advanced illness

Both are composite tools combining performance status and symptoms/labs to estimate prognosis.

2. Anticipatory prescribing refers to:

A. Prescribing antibiotics prophylactically
B. Pre-emptively prescribing medications likely needed in the dying phase, before symptoms arise
C. Prescribing chemotherapy early
D. A legal requirement before hospice admission

Answer: B. Pre-emptively prescribing medications likely needed in the dying phase, before symptoms arise

Anticipatory prescribing ensures drugs for pain, agitation, nausea, and secretions are available without delay in the dying phase.

3. In the last days of life, the preferred route for medication when oral intake fails is:

A. Intramuscular
B. Subcutaneous (often via syringe driver)
C. Rectal only
D. Sublingual only

Answer: B. Subcutaneous (often via syringe driver)

Continuous subcutaneous infusion (syringe driver) is standard when the oral route is no longer feasible.

4. Kerala's Neighbourhood Network in Palliative Care is notable for:

A. Being a purely hospital-based model
B. A community-owned, volunteer-driven home-based model achieving high population coverage
C. Being limited to cancer patients only
D. Being funded exclusively by central government

Answer: B. A community-owned, volunteer-driven home-based model achieving high population coverage

It is a widely cited, scalable, low-cost, community-driven model of home-based palliative care.

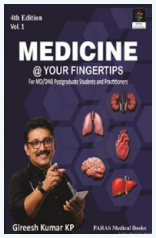
5. Artificial nutrition and hydration in the actively dying patient generally:

A. Reliably prolongs life and improves comfort
B. Confers no proven benefit and may increase discomfort/secretions
C. Is mandatory in all patients
D. Cures the underlying terminal illness

Answer: B. Confers no proven benefit and may increase discomfort/secretions

Artificial nutrition/hydration in the actively dying phase does not improve outcomes and may add burden (e.g. increased secretions).

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MCQ QUESTION BANK · MCQ 12

Mixed Review — Pain and Palliative Medicine

Read the text ›

1. Breakthrough pain rescue doses are typically what fraction of the total 24-h opioid dose?

A. 1–2%
B. 1/6 (≈ 17%)
C. 50%
D. 100%

Answer: B. 1/6 (≈ 17%)

1/6 of the 24-h dose (= the 4-hourly dose) is the IAPC rescue dose.

2. End-of-dose failure of analgesia is best managed by:

A. Adding more rescue/breakthrough doses only
B. Increasing the regular background opioid dose
C. Switching to a non-opioid
D. Stopping all analgesia

Answer: B. Increasing the regular background opioid dose

End-of-dose failure reflects inadequate baseline control and requires increasing the background dose, not just rescue dosing.

3. Pseudoaddiction is best described as:

A. True opioid use disorder requiring addiction treatment
B. Drug-seeking behaviour from undertreated pain that resolves with adequate analgesia
C. A permanent personality trait
D. An indication to stop all opioids immediately

Answer: B. Drug-seeking behaviour from undertreated pain that resolves with adequate analgesia

Pseudoaddiction resolves once pain is adequately controlled, distinguishing it from true opioid use disorder.

4. Methadone rotation should be performed:

A. By any physician using standard equianalgesic tables
B. Only by or with specialist/consultant guidance given its non-linear conversion and long half-life
C. Only in the inpatient ICU setting
D. Never, as it is contraindicated in rotation

Answer: B. Only by or with specialist/consultant guidance given its non-linear conversion and long half-life

Methadone's complex, dose-dependent pharmacokinetics necessitate specialist-level rotation protocols.

5. The WHO ladder principle 'by the clock' refers to:

A. Giving analgesia only when pain occurs
B. Giving regular, scheduled analgesia rather than purely on-demand dosing
C. Timing doses to coincide with meals only
D. Avoiding night-time dosing

Answer: B. Giving regular, scheduled analgesia rather than purely on-demand dosing

Regular scheduled dosing maintains steady analgesic levels rather than reactive, as-needed dosing alone.

MCQ QUESTION BANK · MCQ 13

Infections in the Palliative Care Patient

1. The first dose of antibiotic in suspected neutropenic sepsis should be given within:

A. 15 minutes
B. 1 hour
C. 4 hours
D. After the blood count is available

Answer: B. 1 hour

Time to antibiotic within 1 hour of presentation is the key quality standard; do not wait for the neutrophil count.

2. A MASCC score of ≥ 21 indicates:

A. High risk of complications
B. Low risk — suitable for oral outpatient therapy
C. Need for antifungal therapy
D. Need for G-CSF

Answer: B. Low risk — suitable for oral outpatient therapy

MASCC ≥ 21 identifies low-risk febrile neutropenia suitable for oral antibiotics with close follow-up.

3. PJP prophylaxis is indicated with prednisolone at a dose of at least:

A. 5 mg/day for 1 week
B. 20 mg/day for ≥ 4 weeks
C. 10 mg/day for 3 days
D. Any dose

Answer: B. 20 mg/day for ≥ 4 weeks

≥ 20 mg prednisolone (or equivalent) for ≥ 4 weeks — includes dexamethasone for brain metastases.

4. Black necrotic eschar on the palate of a diabetic neutropenic patient suggests:

A. Aspergillosis
B. Mucormycosis
C. Candidiasis
D. HSV

Answer: B. Mucormycosis

Rhino-orbito-cerebral mucormycosis — liposomal amphotericin B and urgent debridement.

5. Which drug most reduces opioid levels and may precipitate a pain crisis?

A. Isoniazid
B. Rifampicin
C. Ethambutol
D. Pyrazinamide

Answer: B. Rifampicin

Rifampicin induces CYP3A4 and other enzymes, lowering methadone, fentanyl and oxycodone levels.

MCQ QUESTION BANK · MCQ 14

Infections — Diagnosis and Treatment

1. Before rituximab, HBV screening should include:

A. HBsAg only
B. HBsAg and anti-HBc
C. Anti-HBs only
D. HBV DNA only

Answer: B. HBsAg and anti-HBc

Anti-HBc identifies past infection at risk of reactivation even if HBsAg is negative.

2. The best treatment for acute cholangitis due to a blocked stent is:

A. Antibiotics alone
B. Biliary drainage plus antibiotics
C. Steroids
D. Lactulose

Answer: B. Biliary drainage plus antibiotics

Drainage (stent exchange / PTBD) is definitive; antibiotics alone are insufficient.

3. Which is NOT an indication to remove a central line in CRBSI?

A. S. aureus bacteraemia
B. Candidaemia
C. Tunnel infection

D. Uncomplicated coagulase-negative staphylococcal infection

Answer: D. Uncomplicated coagulase-negative staphylococcal infection

CoNS infection can often be treated with line retention plus antibiotic lock.

4. Rasburicase is contraindicated in:

A. Diabetes
B. G6PD deficiency
C. Renal failure
D. Pregnancy

Answer: B. G6PD deficiency

Hydrogen peroxide generated by rasburicase causes haemolysis in G6PD deficiency.

5. Asymptomatic bacteriuria in a long-term catheterised patient should be:

A. Treated with 7 days of antibiotics
B. Not treated
C. Treated with bladder washouts
D. Treated with prophylactic antibiotics

Answer: B. Not treated

Bacteriuria is universal with long-term catheters; treat only symptomatic infection.

MCQ QUESTION BANK · MCQ 15

Electrolyte and Metabolic Disorders I

1. The maximum recommended rise in sodium in chronic hyponatraemia in 24 h is about:

A. 5 mmol/L
B. 10 mmol/L
C. 20 mmol/L
D. 25 mmol/L

Answer: B. 10 mmol/L

Limit correction to ≤ 10 mmol/L (≤ 8 in high-risk) per 24 h to avoid osmotic demyelination.

2. Humoral hypercalcaemia of malignancy is mediated mainly by:

A. Vitamin D
B. PTHrP
C. Calcitonin
D. IGF-2

Answer: B. PTHrP

PTH-related peptide is responsible in ~80% (especially squamous cancers).

3. Calcium gluconate in hyperkalaemia acts by:

A. Shifting K into cells
B. Increasing renal excretion
C. Stabilising the myocardial membrane
D. Binding potassium in the gut

Answer: C. Stabilising the myocardial membrane

It does not lower potassium; insulin–glucose and salbutamol shift K into cells.

4. Hypokalaemia refractory to replacement suggests coexisting:

A. Hypercalcaemia
B. Hypomagnesaemia
C. Hypernatraemia
D. Hypophosphataemia

Answer: B. Hypomagnesaemia

Magnesium deficiency causes renal potassium wasting — correct Mg first.

5. Which drug class commonly causes hypomagnesaemia in oncology?

A. Anti-EGFR antibodies (cetuximab)

B. Bisphosphonates
C. Anthracyclines
D. Taxanes

Answer: A. Anti-EGFR antibodies (cetuximab)

Cetuximab and panitumumab block renal Mg reabsorption; cisplatin also causes wasting.

MCQ QUESTION BANK · MCQ 16

Electrolyte and Metabolic Disorders II

1. Non-islet-cell tumour hypoglycaemia is mediated by:

A. Insulin
B. Big IGF-2
C. Glucagon
D. Cortisol

Answer: B. Big IGF-2

Large mesenchymal or hepatic tumours secrete incompletely processed IGF-2; insulin is suppressed.

2. In type 1 diabetes in the last days of life:

A. Stop all insulin
B. Continue some basal insulin
C. Switch to metformin
D. Check glucose 4-hourly

Answer: B. Continue some basal insulin

Basal insulin prevents distressing DKA; reduce the dose and minimise testing.

3. Sudden collapse with hyponatraemia, hypoglycaemia and hypotension after stopping long-term dexamethasone suggests:

A. SIADH
B. Adrenal crisis
C. Septic shock only
D. Hypothyroidism

Answer: B. Adrenal crisis

HPA-axis suppression → adrenal crisis; give hydrocortisone 100 mg IV.

4. Type B lactic acidosis in lymphoma is due to:

A. Hypoperfusion
B. Warburg effect (aerobic glycolysis)
C. Metformin only
D. Diarrhoea

Answer: B. Warburg effect (aerobic glycolysis)

Tumour aerobic glycolysis produces lactate without hypoperfusion; treat the malignancy.

5. Parenteral hydration (1 L/day) in advanced cancer patients in an RCT (Bruera 2013):

A. Improved survival
B. Reduced delirium significantly
C. Did not improve symptoms or survival
D. Increased hospitalisation

Answer: C. Did not improve symptoms or survival

No benefit over placebo in symptoms, quality of life or survival.

MCQ QUESTION BANK · MCQ 17

Thrombosis, Bleeding and Emergencies

1. Which anticoagulant was shown non-inferior to dalteparin in cancer VTE without excess major bleeding (Caravaggio)?

A. Warfarin
B. Apixaban
C. Edoxaban
D. Fondaparinux

Answer: B. Apixaban

Caravaggio (2020) — apixaban.

2. A Khorana score point is given for platelets of at least:

A. 150 000/µL
B. 250 000/µL
C. 350 000/µL
D. 450 000/µL

Answer: C. 350 000/µL

Pre-chemotherapy platelets ≥ 350 000/µL score 1 point.

3. The first-line drug for a catastrophic terminal haemorrhage is:

A. IV morphine
B. Midazolam 10 mg buccal/IM
C. Tranexamic acid IV
D. Haloperidol

Answer: B. Midazolam 10 mg buccal/IM

Midazolam for distress; staying with the patient and dark towels are equally important.

4. Which should be avoided in cardiac tamponade?

A. IV fluids
B. Pericardiocentesis
C. Diuretics
D. Echocardiography

Answer: C. Diuretics

Diuretics and vasodilators reduce preload and worsen haemodynamics.

5. Treatment of hyperviscosity syndrome due to IgM paraprotein starts with:

A. Red-cell transfusion
B. Plasmapheresis
C. Diuretics
D. Steroids alone

Answer: B. Plasmapheresis

Plasmapheresis rapidly reduces IgM; avoid transfusion before viscosity falls.

MCQ QUESTION BANK · MCQ 18

Algorithms — Pain

1. Rescue (breakthrough) opioid dose is usually:

A. 1/2 of the 24-h dose
B. 1/6 of the 24-h dose
C. Equal to the 4-hourly dose × 2
D. 1/20 of the 24-h dose

Answer: B. 1/6 of the 24-h dose

Equal to the regular 4-hourly dose (IAPC).

2. When rotating opioids because of toxicity, the calculated equianalgesic dose is reduced by:

A. 0%
B. 25–50%
C. 75%

D. 100%

Answer: B. 25–50%

To allow for incomplete cross-tolerance.

3. Converting oral morphine 60 mg/24 h to a syringe driver, the SC morphine dose is:

A. 60 mg
B. 30 mg
C. 20 mg
D. 120 mg

Answer: B. 30 mg

Oral:SC morphine ratio is 2:1.

4. Which opioid is safest in eGFR < 10 mL/min?

A. Morphine
B. Codeine
C. Fentanyl
D. Tramadol

Answer: C. Fentanyl

Fentanyl, alfentanil and buprenorphine lack active renally excreted metabolites.

5. Coeliac plexus neurolysis is most useful for pain from cancer of the:

A. Rectum
B. Pancreas
C. Cervix
D. Lung

Answer: B. Pancreas

Upper abdominal visceral pain (pancreas, stomach).

MCQ QUESTION BANK · MCQ 19

Algorithms — Symptoms

1. The antiemetic of choice for chemically induced nausea (e.g. opioid, hypercalcaemia) is:

A. Cyclizine
B. Haloperidol
C. Metoclopramide
D. Hyoscine

Answer: B. Haloperidol

Haloperidol blocks D2 receptors at the chemoreceptor trigger zone.

2. Metoclopramide should be avoided in:

A. Gastric stasis
B. Complete bowel obstruction with colic
C. Opioid-induced nausea
D. Hepatomegaly

Answer: B. Complete bowel obstruction with colic

Prokinetics worsen colic in complete obstruction.

3. Octreotide in malignant bowel obstruction acts by:

A. Increasing peristalsis
B. Reducing gastrointestinal secretions
C. Blocking D2 receptors
D. Relieving constipation

Answer: B. Reducing gastrointestinal secretions

Somatostatin analogue reduces secretions and vomiting volume.

4. The most evidence-based non-drug measure for breathlessness is:

A. Oxygen for all

B. Hand-held fan to the face
C. Bed rest
D. Nebulised saline

Answer: B. Hand-held fan to the face

Facial airflow reduces the sensation of breathlessness.

5. Anticholinergics for death rattle work by:

A. Clearing existing secretions
B. Reducing production of new secretions
C. Sedating the patient
D. Bronchodilation

Answer: B. Reducing production of new secretions

Hence start early; positioning and explanation are key.

MCQ QUESTION BANK · MCQ 20

Algorithms — Emergencies and End of Life

1. Initial dexamethasone dose in suspected spinal cord compression is:

A. 4 mg/day
B. 16 mg/day
C. 100 mg bolus always
D. 1 mg/day

Answer: B. 16 mg/day

16 mg/day with gastroprotection; urgent MRI within 24 h.

2. In SVCO with suspected lymphoma, steroids should ideally be given:

A. Immediately before biopsy
B. After obtaining histology
C. Never
D. Only intrathecally

Answer: B. After obtaining histology

Steroids can obscure the histological diagnosis of lymphoma.

3. Naloxone in opioid toxicity in a cancer patient should be given:

A. 400 µg IV bolus
B. In 100 µg increments titrated to respiration
C. Orally
D. Only if pupils are dilated

Answer: B. In 100 µg increments titrated to respiration

Small titrated doses avoid acute withdrawal and severe pain.

4. In proportionate palliative sedation, the first-line drug is:

A. Phenobarbital
B. Midazolam
C. Propofol
D. Morphine

Answer: B. Midazolam

Midazolam titrated to comfort; opioids are not sedatives for this purpose.

5. In India, withdrawal of life support for an incapacitated patient with a living will requires:

A. Only family consent
B. Primary and Secondary Medical Boards with JMFC informed
C. High Court approval
D. Police permission

Answer: B. Primary and Secondary Medical Boards with JMFC informed

Common Cause 2018 as simplified in 2023.

MCQ QUESTION BANK · MCQ 21

Prognostication and Communication

1. A Palliative Prognostic Index (PPI) > 6 predicts survival of less than:

A. 3 days
B. 3 weeks
C. 6 months
D. 1 year

Answer: B. 3 weeks

PPI > 6 predicts survival < 3 weeks.

2. The 'surprise question' asks whether you would be surprised if the patient died within:

A. 1 week
B. 1 month
C. 12 months
D. 5 years

Answer: C. 12 months

A 'no' answer identifies patients who may benefit from palliative care.

3. In SPIKES, the 'I' stands for:

A. Information
B. Invitation
C. Intervention
D. Introduction

Answer: B. Invitation

Invitation — ask how much the patient wishes to know.

4. NURSE statements are used to respond to:

A. Prognosis
B. Emotions
C. Drug doses
D. Legal issues

Answer: B. Emotions

Name, Understand, Respect, Support, Explore.

5. Clinicians' survival estimates are typically:

A. Accurate
B. Overoptimistic
C. Pessimistic
D. Random

Answer: B. Overoptimistic

Overestimated by a factor of 3–5 (Christakis).

MCQ QUESTION BANK · MCQ 22

Special Populations and Non-Malignant Disease

1. WHO 2012 guidelines on persisting pain in children recommend:

A. A three-step ladder with codeine
B. A two-step approach
C. Tramadol first line
D. No opioids under 12 years

Answer: B. A two-step approach

Paracetamol/ibuprofen, then a strong opioid (morphine); codeine and tramadol avoided.

2. PEG feeding in advanced dementia:

A. Prolongs survival
B. Prevents aspiration
C. Has not been shown to prolong survival or prevent aspiration
D. Heals pressure ulcers

Answer: C. Has not been shown to prolong survival or prevent aspiration

Careful hand feeding is recommended.

3. The preferred opioid for pain in a patient who has stopped dialysis is:

A. Morphine
B. Fentanyl or alfentanil
C. Codeine
D. Pethidine

Answer: B. Fentanyl or alfentanil

Renal-safe opioids without active renally cleared metabolites.

4. PAINAD is used to assess pain in:

A. Children < 3 years
B. Patients with advanced dementia
C. ICU ventilated patients only
D. Cognitively intact adults

Answer: B. Patients with advanced dementia

Five observational items scored 0–2 each.

5. Gabapentin for uraemic pruritus in haemodialysis is usually dosed:

A. 600 mg tds
B. 100–300 mg after dialysis
C. 1200 mg daily
D. Not used

Answer: B. 100–300 mg after dialysis

Low dose after each dialysis session because of renal clearance.

MCQ QUESTION BANK · MCQ 23

Treatment-Related Complications

1. The NK1 antagonist used for delayed CINV is:

A. Ondansetron
B. Aprepitant
C. Metoclopramide
D. Domperidone

Answer: B. Aprepitant

Aprepitant blocks substance P at NK1 receptors.

2. The only drug with good RCT evidence for painful CIPN is:

A. Gabapentin
B. Duloxetine
C. Amitriptyline
D. Acetyl-L-carnitine

Answer: B. Duloxetine

Duloxetine (Smith et al., JAMA 2013); ASCO recommends it.

3. Cold-induced acute dysaesthesia is characteristic of:

A. Paclitaxel
B. Oxaliplatin
C. Vincristine

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D. Bortezomib

Answer: B. Oxaliplatin

Oxaliplatin acute neurotoxicity is triggered by cold.

4. Radiation pneumonitis typically presents:

A. During the first week of RT
B. 1–6 months after thoracic RT
C. Years later only
D. Immediately after a single fraction

Answer: B. 1–6 months after thoracic RT

Treated with tapering prednisolone.

5. Cumulative doxorubicin dose above which cardiomyopathy risk rises steeply:

A. 100 mg/m²
B. 250 mg/m²
C. 450 mg/m²
D. 1000 mg/m²

Answer: C. 450 mg/m²

Risk increases markedly above 400–450 mg/m².

MCQ QUESTION BANK · MCQ 24

Metabolic and Endocrine Complications

1. In combined cortisol and thyroid deficiency, the first replacement should be:

A. Levothyroxine
B. Hydrocortisone
C. Fludrocortisone
D. Either

Answer: B. Hydrocortisone

Thyroxine first can precipitate adrenal crisis.

2. Checkpoint-inhibitor thyroiditis usually begins with:

A. Hypothyroidism
B. Transient thyrotoxicosis
C. Thyroid storm
D. Goitre

Answer: B. Transient thyrotoxicosis

Thyrotoxic phase followed by hypothyroidism.

3. Valproate-induced hyperammonaemia is treated with:

A. Rifaximin
B. Levocarnitine
C. Insulin
D. Calcitonin

Answer: B. Levocarnitine

Levocarnitine plus stopping valproate.

4. For nausea in uraemia, the preferred antiemetic is:

A. High-dose metoclopramide
B. Low-dose haloperidol
C. Cyclizine 150 mg
D. Domperidone 40 mg

Answer: B. Low-dose haloperidol

Haloperidol 0.5–1 mg; avoid accumulating drugs.

5. In SBP, albumin is given to:

A. Treat infection

B. Reduce renal failure and mortality
C. Correct sodium
D. Reduce ascites permanently

Answer: B. Reduce renal failure and mortality

1.5 g/kg day 1 and 1 g/kg day 3 reduces hepatorenal syndrome.

MCQ QUESTION BANK · MCQ 25

Malignant Spinal Cord Compression and Bowel Obstruction

1. The strongest predictor of walking after treatment of MSCC is:

A. Tumour type
B. Ambulatory status at diagnosis
C. Age
D. Steroid dose

Answer: B. Ambulatory status at diagnosis

Hence urgent recognition and imaging.

2. Imaging of choice in suspected MSCC is:

A. Plain X-ray spine
B. Whole-spine MRI
C. Bone scan
D. CT abdomen

Answer: B. Whole-spine MRI

Whole spine — multilevel disease is common.

3. In malignant bowel obstruction, octreotide's main benefit is:

A. Relief of colic
B. Reduction in vomiting volume
C. Treatment of obstruction
D. Laxative effect

Answer: B. Reduction in vomiting volume

Antisecretory action.

4. Which is contraindicated in complete MBO with colic?

A. Haloperidol
B. Metoclopramide
C. Hyoscine butylbromide
D. Dexamethasone

Answer: B. Metoclopramide

Prokinetics worsen colic.

5. A single 8 Gy fraction for painful bone metastasis is:

A. Inferior to multiple fractions for pain
B. Equivalent for pain relief
C. Contraindicated
D. Only for spine

Answer: B. Equivalent for pain relief

Equivalent pain relief; retreatment slightly more frequent.

MCQ QUESTION BANK · MCQ 26

Infections and Fever in India

1. Pleural fluid ADA > 40 U/L in an Indian patient suggests:

A. Malignant effusion

B. Tuberculous effusion
C. Empyema
D. Transudate

Answer: B. Tuberculous effusion

High ADA supports TB pleuritis; confirm with biopsy/CBNAAT.

2. A febrile patient with an eschar and pneumonitis most likely has:

A. Dengue
B. Scrub typhus
C. Malaria
D. Leptospirosis

Answer: B. Scrub typhus

Treat with doxycycline.

3. In possible dengue, the antipyretic of choice is:

A. Ibuprofen
B. Aspirin
C. Paracetamol
D. Diclofenac

Answer: C. Paracetamol

NSAIDs increase bleeding risk with thrombocytopenia.

4. Severe malaria is treated first with:

A. Chloroquine
B. IV artesunate
C. Primaquine
D. Doxycycline alone

Answer: B. IV artesunate

Artesunate 2.4 mg/kg at 0, 12, 24 h then daily.

5. Ceftazidime–avibactam with aztreonam is used for:

A. MRSA
B. NDM-producing CRE
C. C. difficile
D. VRE

Answer: B. NDM-producing CRE

Aztreonam is stable to metallo-β-lactamases; avibactam protects it from co-produced enzymes.

MCQ QUESTION BANK · MCQ 27

Bleeding, Breathlessness and Mouth Care

1. Tranexamic acid should be used cautiously in:

A. Haemoptysis
B. Haematuria
C. Fungating wound bleeding
D. Rectal bleeding

Answer: B. Haematuria

Risk of clot retention in the urinary tract.

2. Lymphangitis carcinomatosa is best initially treated with:

A. Antibiotics
B. Dexamethasone trial
C. Diuretics
D. Pleurodesis

Answer: B. Dexamethasone trial

Steroid trial plus opioid for breathlessness.

3. Pilocarpine helps xerostomia only if:

A. The patient is dehydrated
B. Residual salivary gland function exists
C. Given IV
D. Candida is present

Answer: B. Residual salivary gland function exists

It stimulates remaining glandular tissue.

4. Which mouthwash is used for mucositis pain?

A. Chlorhexidine
B. Benzydamine
C. Hydrogen peroxide
D. Alcohol-based

Answer: B. Benzydamine

Benzydamine is anti-inflammatory and analgesic.

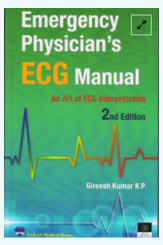
5. Weight gain with megestrol acetate is mainly:

A. Lean muscle
B. Fat and fluid
C. Bone
D. Tumour

Answer: B. Fat and fluid

Hence it improves appetite but not function.

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SECTION 5

Landmark Trials in Palliative Medicine & Oncology

A compact, verified reference of the trials and policy documents most often invoked in viva and essay answers – each citation links directly to its real primary source so you can read the original abstract or guideline.

5.1 Early Palliative Care Integration 424

5.2 Symptom & Complication Management 425

AT A GLANCE

Landmark evidence at a glance

Theme	Key trial
Early palliative care	Temel 2010 (NEJM), ENABLE III, Zimmermann 2014
Breathlessness	Abernethy 2010 (oxygen vs air), fan studies
Delirium	Agar 2017, Hui 2017
Hydration	Bruera 2013
Cord compression	Patchell 2005
Cancer VTE	CLOT 2003, Caravaggio 2020

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5.1 Early Palliative Care Integration

These three randomised/cluster-randomised trials form the evidence base for early, concurrent palliative care referral in oncology – one of the most frequently asked themes across Paper 2 long-essay and viva questions.

1. Temel JS, et al. Early Palliative Care for Patients with Metastatic Non–Small-Cell Lung Cancer

CITATION

Temel JS, Greer JA, Muzikansky A, et al. *N Engl J Med* 2010;363:733–742. PubMed: PMID 20818875

KEY FINDING & PRACTICAL TAKEAWAY

RCT (n=151) of early palliative care integrated with standard oncology care vs standard care alone in newly diagnosed metastatic NSCLC. Early palliative care improved quality of life and mood, led to less aggressive end-of-life care, and – despite less aggressive care – was associated with **longer median survival** (11.6 vs 8.9 months). The single most-cited trial justifying early (not just end-stage) palliative care referral.

2. Zimmermann C, et al. Early Palliative Care for Patients with Advanced Cancer: A Cluster-Randomised Controlled Trial

CITATION

Zimmermann C, Swami N, Krzyzanowska M, et al. *Lancet* 2014;383:1721–1730. Lancet abstract (DOI: 10.1016/S0140-6736(13)62676-8)

KEY FINDING & PRACTICAL TAKEAWAY

Cluster-RCT across 24 oncology clinics (n=461) comparing early palliative care intervention with usual care in patients with advanced cancer (not limited to lung cancer). Showed significant improvement in quality of life and satisfaction with care at 3 months, with the benefit becoming more pronounced over time – supporting early referral across tumour types, not only NSCLC.

3. Bakitas MA, et al. ENABLE III – Early versus Delayed Initiation of Concurrent Palliative Oncology Care

CITATION

Bakitas MA, Tosteson TD, Li Z, et al. *J Clin Oncol* 2015;33:1438–1445. ASCO Publications (DOI: 10.1200/JCO.2014.58.6362)

KEY FINDING & PRACTICAL TAKEAWAY

RCT comparing early (within 30–60 days of diagnosis) vs delayed (3 months later) initiation of a nurse-led telephone-based palliative care intervention (ENABLE model) in advanced cancer. One-year survival was significantly better with early initiation (63% vs 48%); supports the 'earlier is better' principle for structured palliative care programmes, including non-physician-delivered models.

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5.2 Symptom & Complication Management

These trials and the foundational WHO guideline are the evidence base most often quoted for specific symptom-control and complication-management decisions in the theory and viva papers.

4. WHO Guidelines for the Pharmacological and Radiotherapeutic Management of Cancer Pain

CITATION

World Health Organization. Geneva: WHO; 2018. WHO publication 9789241550390

KEY FINDING & PRACTICAL TAKEAWAY

The 2018 revision of the original 1986 WHO analgesic ladder guideline. Retains the core principles – oral, by the clock, individualised dosing – while removing the rigid three-step 'ladder' sequencing in favour of choosing the starting step by pain severity. Essential primary-source reading: almost every exam question on the 'WHO analgesic ladder' is really asking about this document.

5. Lee AYY, et al. The CLOT Trial – Low-Molecular-Weight Heparin versus a Coumarin for Cancer-Associated VTE

CITATION

Lee AYY, Levine MN, Baker RI, et al. *N Engl J Med* 2003;349:146–153. PubMed: PMID 12853587

KEY FINDING & PRACTICAL TAKEAWAY

RCT (n=676) establishing that dalteparin (LMWH) was superior to warfarin for preventing recurrent VTE in cancer patients, without increased bleeding. This trial made LMWH (rather than a vitamin-K antagonist) the long-standing standard for cancer-associated thrombosis, now partly superseded by DOAC trials (see below).

6. Agnelli G, et al. The CARAVAGGIO Trial – Apixaban versus Dalteparin for Cancer-Associated VTE

CITATION

Agnelli G, Becattini C, Meyer G, et al. *N Engl J Med* 2020;382:1599–1607. PubMed: PMID 32223112

KEY FINDING & PRACTICAL TAKEAWAY

RCT (n=1155) showing the oral DOAC apixaban was non-inferior to dalteparin for treatment of cancer-associated VTE, without the increased GI bleeding seen with some other DOACs in cancer populations. Key modern evidence behind using an oral anticoagulant instead of daily LMWH injections in suitable patients.

7. Breitbart W, et al. Haloperidol, Chlorpromazine and Lorazepam in Delirium

CITATION

Breitbart W, Marotta R, Platt MM, et al. *Am J Psychiatry* 1996;153:231–237. PubMed: PMID 8561204

KEY FINDING & PRACTICAL TAKEAWAY

Double-blind RCT in hospitalised AIDS patients comparing haloperidol, chlorpromazine and lorazepam for delirium. Both antipsychotics improved delirium; lorazepam monotherapy was ineffective and caused treatment-limiting sedation/worsening cognitive impairment – foundational evidence against benzodiazepine monotherapy for non-withdrawal-related delirium, still quoted in current guidelines.

EXAM-ORIENTED THEORY

More trials every postgraduate should know

Trial	Key message
Bruera 2013 (hydration)	1 L/day parenteral hydration did not improve symptoms or survival in advanced cancer

Trial	Key message
Abernethy 2010 (oxygen vs air)	Palliative oxygen no better than medical air for non-hypoxaemic breathlessness
Patchell 2005 (MSCC)	Surgery + RT better than RT alone for ambulation in selected MSCC
Caravaggio 2020	Apixaban non-inferior to dalteparin for cancer VTE
Hui 2017 (agitated delirium)	Haloperidol + lorazepam reduced agitation vs haloperidol alone in the last days
Agar 2017	Risperidone/haloperidol worsened mild–moderate delirium symptoms vs placebo (non-terminal)
QUARTZ 2016	WBRT added little for poor-prognosis NSCLC brain metastases
Bandieri 2016	Low-dose morphine better than weak opioids for moderate cancer pain
Stopeck 2010	Denosumab delayed skeletal events more than zoledronate in breast cancer

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SECTION 6

DNB Previous-Year Question Papers (Official NBE Archive)

Genuine DNB Palliative Medicine theory papers from the National Board of Examinations' own public archive (October 2024 session), given here as paraphrased topic summaries with a direct link to each official PDF.

6.1 Paper 1 – October 2024 **428**

6.2 Paper 2 – October 2024 **429**

6.3 Paper 4 – October 2024 **430**

AT A GLANCE

How to use the DNB papers

Step	Advice
1	Read the whole paper — note the weight of each question
2	Use the 'Read in text ›' link beside each question to revise its topic
3	Plan answers: definition → classification → table → flowchart → recent advance
4	Practise writing each 10-mark answer in 12–14 minutes
5	Draw at least one flowchart or table per answer

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6.1 DNB Palliative Medicine – Paper 1 – October 2024

OFFICIAL SOURCE

Open the official NBE question paper (PDF)

Source: National Board of Examinations (NBE), official DNB Palliative Medicine theory question paper, October 2024 examination session (natboard.edu.in). Section 1, offline, all questions mandatory and subjective; 180 minutes; 100 marks (10 marks per question). Topics below are paraphrased summaries preserving every question and sub-part – open the official PDF (link above) for the exact wording and mark break-up.

1. **Antiemetic physiology and drugs**
   - The pathways that trigger vomiting
   - How chemotherapy agents are grouped by their likelihood of causing nausea and vomiting
   - Medications for nausea and vomiting that do not respond to standard treatment
2. **Stroke and neurology in palliative care**
   - A labelled diagram of the circle of Willis and an account of cerebral blood flow
   - Situations in a stroke patient that should prompt a palliative care referral
   - Factors that guide decisions to limit treatment in advanced stroke
3. **Constipation**
   - Categories of laxatives and other drugs used for constipation
   - Managing constipation caused by opioids
   - Managing constipation in a patient with spinal cord compression
4. **Models of palliative care delivery**
   - The main ways palliative care services are organised and delivered
   - Core principles of home-based palliative care
   - The embedded model of palliative care
5. **Lymphoedema**
   - How lymphoedema develops
   - How lymphoedema is graded by severity
   - The occupational therapist's role in managing it
6. **Pruritus (itch)**
   - The mechanisms that produce itching
   - Itch caused by opioids
   - Itch caused by liver disease
7. **Holistic concepts of suffering** Read in text ›
   - The biopsychosocial model of illness and why it matters in palliative care
   - The concept of total pain
   - Spiritual pain
8. **Mind-body therapies**
   - An overview of mind-body therapies used in palliative care
   - How these therapies may relieve symptoms
   - One mind-body therapy with supporting evidence, described in detail 9. **Addiction in palliative care**
   - Addiction as it applies to palliative care
   - The biological mechanisms and risk factors for opioid addiction
   - Strategies to manage and prevent opioid addiction
10. **Hiccups**
   - The mechanisms behind persistent hiccups in palliative patients
   - How to tell apart hiccup episodes, persistent hiccups, and intractable hiccups
   - Managing intractable hiccups

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6.2 DNB Palliative Medicine – Paper 2 – October 2024

OFFICIAL SOURCE

Open the official NBE question paper (PDF)

Source: National Board of Examinations (NBE), official DNB Palliative Medicine theory question paper, October 2024 examination session (natboard.edu.in). Section 1, offline, all questions mandatory and subjective; 180 minutes; 100 marks (10 marks per question). Topics below are paraphrased summaries preserving every question and sub-part – open the official PDF (link above) for the exact wording and mark break-up.

1. **Frailty and older adults with cancer**
   - Pathophysiology and clinical features of frailty
   - Comprehensive geriatric assessment in older adults with cancer
   - Principles of geriatric palliative care in oncology
2. **Malignant pleural effusion** Read in text ›
   - Pathophysiology
   - Pleural fluid analysis
   - Agents used for pleurodesis
3. **Methadone in cancer pain**
   - Its role
   - Drug interactions
   - Prescribing and monitoring principles
4. **Palliative care trajectories and upstreaming**
   - Newer illness trajectories
   - Evidence for early palliative care integration in oncology
   - Strategies to enable it
5. **Financial toxicity in cancer care** Read in text ›
   - Description
   - Assessment tools
   - Management in oncology settings
6. **Pain after cancer treatment**
   - Chronic pain syndromes linked to antineoplastic therapy
   - G-CSF-associated bone pain
   - Midline retroperitoneal syndrome
7. **Xerostomia in head and neck cancer** Read in text ›
   - Causes
   - Clinical assessment
   - Management
8. **Lymphangitis carcinomatosa** Read in text ›
   - Causes
   - Clinical presentation
   - Management 9. **Malignant bowel obstruction**
   - Pathophysiology
   - Approach to recurrent obstruction
   - Conservative management of recurrent obstruction
10. **Survivorship in cancer**
    - Survivorship care for patients with advanced cancer
    - Comprehensive care needs of cancer survivors
    - Role of palliative care in survivorship

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6.3 DNB Palliative Medicine – Paper 4 – October 2024

OFFICIAL SOURCE

Open the official NBE question paper (PDF)

Source: National Board of Examinations (NBE), official DNB Palliative Medicine theory question paper, October 2024 examination session (natboard.edu.in). Section 1, offline, all questions mandatory and subjective; 180 minutes; 100 marks (10 marks per question). Topics below are paraphrased summaries preserving every question and sub-part – open the official PDF (link above) for the exact wording and mark break-up.

1. **Pressure sores**
   - How to assess a patient's risk of developing them
   - How they are classified by stage
   - Evidence-based guidance on preventing and treating them
2. **Breakthrough cancer pain**
   - Its defining characteristics
   - Basic principles of drug treatment
   - The evidence supporting its management
3. **Sleep problems in palliative care**
   - What causes sleep disturbance in these patients
   - Sleep hygiene practices
   - Evidence-based management of sleep issues
4. **Quality-Adjusted Life Years (QALYs)**
   - What the measure means
   - How it applies in palliative care settings
   - Evidence on its use in oncology
5. **Complex interventions in palliative care** Read in text ›
   - What defines a complex intervention
   - The Medical Research Council's steps for developing one
   - Three published palliative care examples
6. **Idiopathic pulmonary fibrosis (IPF)**
   - How the disease typically progresses
   - Factors guiding when to limit treatment in ICU
   - The role of palliative care in IPF
7. **India's opioid availability** Read in text ›
   - Main amendments to the NDPS Act
   - Current status of opioid availability in India
   - How those amendments have improved access
8. **Trial reporting and appraisal**
   - A brief overview of the CONSORT guidelines
   - How CONSORT helps critically appraise randomised controlled trials
   - Fast-track trials in palliative care 9. **Needs assessment**
   - The Supportive and Palliative Care Indicators Tool (SPICT)
   - How SPICT supports palliative care needs assessment
   - Indian evidence on needs assessment
10. **Interventional radiology in palliative care**
    - Its role in managing these patients
    - Two procedures commonly used
    - Ethical dilemmas in deciding whether to offer or withhold such procedures

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APPENDIX

NMC CBME Curriculum

The National Medical Commission's Guidelines for Competency Based Postgraduate Training Programme for MD in Palliative Medicine, reproduced in full and organised for quick revision — objectives, competencies, the complete ten-section syllabus (Cognitive, Affective and Psychomotor domains), teaching–learning methods, the assessment scheme and recommended reading. Pain Management is listed here exactly as the NMC syllabus places it (Section CD3.1); its full examination-ready treatment — definitions, drug tables, pearls — follows separately in Section 3 (Pain Management).

1 Preamble, Objectives & Competencies 431

2 Syllabus — Cognitive Domain (CD1–CD10) 433

3 Syllabus — Affective Domain (AD1–AD10) 441

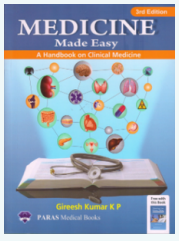
4 Syllabus — Psychomotor Domain (PD1–PD10) 444

5 Teaching & Learning Methods 446

6 Assessment Scheme 447

7 Recommended Reading & Appraisal Form 448

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1. Preamble, Objectives & Competencies

Preamble

Palliative Medicine is a broad medical specialty that involves the study and management of patients with active, progressive, far-advanced disease, for whom the prognosis is limited and the goal and focus of care is relief of symptoms and quality of life. The Indian Association of Palliative Care (IAPC) defines Palliative Care (Medicine) as "the active total care applicable from the time of diagnosis, aimed at improving the quality of life of patients and their families facing a serious life-limiting illness, through the prevention and relief of suffering from pain and other physical symptoms as well as psychological, social and spiritual distress, through socially acceptable and affordable interventions."

Key features: recognition and relief of pain and other symptoms; recognition and relief of psychosocial suffering, including care and support for families and caregivers; recognition and relief of spiritual/existential suffering; recognition of End of Life Care needs and provision of End of Life Care and bereavement support after death. Palliative Medicine applies to all life-limiting conditions — cancer, advanced HIV/AIDS, end-stage organ failure, chronic neurodegenerative conditions and more — and should be applied early and integrated with all health services.

Specialist training involves 24 months of broad (Core) experience in Palliative Medicine and 12 months of focused (Non-Core) experience in cancer medicine, general medicine and related subspecialties.

Subject-specific objectives (14)

- Manage pain and other physical symptoms, using appropriate clinical assessment methods, rational investigations and relief by pharmacological and non-pharmacological methods. p.397 p.393
- Explain the role of psychological, emotional, social, spiritual and existential issues in illness, suffering and symptom manifestation, taking into account the socio-cultural context of the patient and family. p.239 p.267
- Manage issues in illness, suffering and symptom manifestation clinically, by self, with multidisciplinary help, and by referral to relevant specialists. p.357 p.346
- Provide good supportive care in patients with advanced life-limiting illness and manage concurrent illness, complications, co-morbid illness and emergencies. p.316 p.39
- Provide specialist palliative care in all clinical settings — outpatients, ward, home, hospice and consultation liaison. p.321 p.369
- Recognise the terminal phase and the dying process; participate in effective end-of-life decision-making; communicate with and plan good end-of-life care with the family. p.288 p.60
- Communicate with the family sensitively and empathically; break bad news; manage difficult and advanced communication situations. p.350 p.280
- Communicate effectively with peers, supervisors and other members of the team. p.357
- Mentor and supervise junior doctors, maintain active interest in academics and exhibit a high level of teaching. p.352
- Undertake research in palliative care — observational studies, RCTs and clinical audits. p.379 p.317
- Incorporate Evidence Based Medicine (EBM) and Good Clinical Practice into patient care and teaching. p.354 p.430
- Manage human resources, finance, quality assurance and data management aspects of a palliative care service. p.409 p.379
- Develop life-long learning skills to keep knowledge and skills in advanced palliative care current. p.379 p.317
- Recognise stress and burn-out, institute mitigation measures, and recognise the need for self-care. p.377

Subject-specific competencies — summary

Domain	Summary Of Expected Competence
A. Cognitive (10 areas)	Literature review and currency; clarity and presentation; engaging audience; feedback evaluation; evidence-based medicine; qualitative/quantitative research; study design and statistics; good clinical practice in research; critical appraisal and scientific writing. p.410 p.430
B. Affective (5 areas)	Working in a multidisciplinary/interdisciplinary team; recognising contributions of other team members; empowering patients/families facing life-limiting illness; recognising stress/burn-out and self-care; supervision, monitoring and leadership. p.357 p.389 p.341

Domain	Summary Of Expected Competence
C. Psychomotor (10 areas)	Comprehensive assessment and management of pain, physical, psychological, spiritual and social issues; communication skills; disease management options (oncology/non-oncology); supportive care; managing co-morbidity and complications; comprehensive end-of-life care; expert clinical decision-making respecting socio-cultural context; ethics-based decision-making; specialist palliative care across all age groups and clinical settings. p.345 p.321 p.299

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2. Syllabus — Cognitive Domain (CD1–CD10)

p.4xx Filled chips open the Section 7 supplement

This Syllabus outlines the broad concepts, learning objectives, theoretical knowledge (Cognitive Domain), attitudes and behaviour (Affective Domain) and clinical skills (Psychomotor Domain) required to become a specialist Palliative Medicine physician. Attaining competency in all aspects of this curriculum is expected to take three years of supervised training.

SECTION CD1: INTRODUCTION TO PALLIATIVE MEDICINE

No.	Topic	Essentials
1.1.1	**History of Palliative Medicine**	• Ancient history of hospice care • Dame Dr. Cicely Saunders and St. Christopher's Hospice • History and philosophy of the Hospice movement • Modern Hospice movement and evolution of palliative care • Evolution of Palliative Medicine • History of Indian Palliative Care movement p.389 p.369 p.399 p.458
1.2.1	**Principles of Palliative Medicine 1**	• Definitions (Palliative Care, Palliative Approach, Palliative Procedure, Generalist and Specialist Palliative Care) • Illness trajectories and stages • Estimating the Palliative Care need • Cardinal concepts underlying the philosophy of palliative medicine • WHO Principles of Palliative Care • Holistic Care p.348 p.399 p.354 p.458 p.481
1.2.2	**Principles of Palliative Medicine 2**	• The 12 WHO/IAHPC principles of palliative care — unit of care includes patient and family; symptoms routinely assessed and managed; ethical decision-making; interdisciplinary team delivery; continuity of care; dying as a normal part of life; attention to spiritual/existential distress; neither hastens nor prolongs dying; bereavement support; preserves well-being of staff; continuous quality improvement and research; advocacy for patients and health policy p.348 p.399 p.389 p.458
1.3.1	**Speciality of Palliative Medicine**	• Levels of Care (Level 1–3) • Development of Palliative Medicine as a speciality • Core competencies of a Palliative Medicine physician • Specialist Palliative Medicine Service • CanMEDS Physician Competency Framework • Avoiding downsides of specialist training p.354 p.321 p.410 p.458
1.4.1	**Multidisciplinary team 1**	• Concept of Shared Care • Multidisciplinary vs interdisciplinary team • Role of nurse, medical social worker, occupational/physiotherapist, clinical psychologist/ counsellor, nutritionist p.357 p.339 p.346
1.4.2	**Multidisciplinary team 2**	• Role of wound/stoma therapist, speech & language specialist, volunteer, chaplain/spiritual-care person, clinical pharmacist, music/art/play therapist, yoga and CAM specialist p.357 p.354 p.339
1.5.1	**Models of Palliative Care Delivery 1**	• Stjernswärd's Palliative Care for All Model; Early, Acute, Integrated, Simultaneous/shared-care models — description, mode of delivery, advantages/ disadvantages, evidence p.352 p.398 p.379
1.5.2	**Models of Palliative Care Delivery 2**	• In-patient PC unit; free-standing hospice; hospital PC team (consultation liaison); community PC (home-based); out-patient PC unit; day PC unit — team composition, scope, skills, staffing, infrastructure, benefits/disadvantages p.352 p.370 p.357
1.6.1	**Research in Palliative Medicine 1**	• Scope, ethics and barriers for research in PM • Evidence based palliative medicine (Oxford CEBM levels of evidence, obtaining evidence, citation databases, judging trial/review quality, critical evaluation of an RCT and systematic review) • Conducting a clinical trial in Palliative Medicine p.410 p.354 p.389
1.6.2	**Research in Palliative Medicine 2**	• Writing a research protocol (identifying the area, clinical problem, literature review, research question, objectives, study design, outcomes, statistics, interpretation, conclusion) • Qualitative research; psycho-social research in Palliative Medicine p.410 p.354 p.389

No.	Topic	Essentials
1.7.1 / 1.7.2	**Scales and tools (1 & 2)**	• Broad multi-symptom assessment scales; performance status; pain; dyspnea; fatigue; delirium; anxiety; depression; distress; spiritual/existential distress; coping/adaptation; social issues; care-giving issues; family issues; communication & satisfaction; sexuality/intimacy; paediatric aspects of advanced illness p.375 p.239 p.243
1.8.1	**Advocacy in Palliative Medicine**	• Policy advocacy (institutional/state/national) • Capacity-building advocacy (resources/funds for infrastructure) • Drug-availability advocacy (access to pain/symptom-control drugs, Essential Medication List) • Education-related advocacy p.395 p.407 p.354 p.475
1.9.1	**Health Policy & Programmes**	• Maharashtra & Kerala State Palliative Care Policy • WHO Palliative Care Collaborating Centres • Network/neighbourhood model in Palliative Care • National Palliative Care Strategy for India • Narcotic Drugs and Psychotropic Substances (NDPS) Act and amendments p.352 p.389 p.329 p.458 p.475
1.10.1	**Quality & Standards**	• Quality and standards in PM; classification/types of standards; country-specific international standards; End of Life Care standards; the Gold Standards Framework; Clinical Practice Guidelines applicable to Palliative Care p.288 p.238 p.369

SECTION CD2: PALLIATIVE PHARMACOLOGY

No.	Topic	Essentials
2.1.1	**Non-steroidal anti-inflammatory drugs**	• COX pathway; classification (COX, efficacy, potency); pharmacokinetics; Type A/B reactions; NSAIDs by organ system (renal, hepatic, CVS, GI, lung, platelets, bone, GU); pharmacology of aspirin, diclofenac, paracetamol, ibuprofen, ketorolac, oxicams, etoricoxib; rational & safe NSAID prescribing p.248 p.247 p.263
2.1.2	**Opioids 1**	• Definitions; opioid receptors; classification (chemical and receptor-based); metabolism and metabolites; pharmacokinetics; use in renal/hepatic impairment; common adverse effects and management; systemic effects of long-term use; opioid-induced respiratory depression and hyperalgesia p.252 p.403 p.263 p.476
2.1.3	**Opioids 2**	• Opioid potency and conversion tables; opioid rotation; pharmacology of weak opioids (codeine, tramadol, tapentadol, dextropropoxyphene) and strong opioids (morphine, fentanyl, buprenorphine, oxycodone, hydromorphone); initiating and titrating strong opioids; instructions to patients and caregivers p.359 p.402 p.297 p.476 p.479
2.1.4	**Adjuvant Analgesics 1 (neuropathic pain)**	• Antidepressants (TCAs, SSRIs); anti-epileptics; anti-arrhythmics (Na-channel blockers); NMDA-receptor antagonists; K-channel openers; GABA-inhibitory/glutamate-excitatory drugs; corticosteroids; Neuropathic Pain Step Ladder p.404 p.362 p.363 p.477
2.1.5	**Adjuvant Analgesics 2**	• Bone pain adjuvants (dexamethasone, calcitonin, bisphosphonates); GI-pain adjuvants (hyoscine, dicyclomine, octreotide); genitourinary-pain adjuvants (oxybutynin, tolterodine, solifenacin, phenazopyridine, propantheline, tamsulosin, flavoxate); myofascial-pain/spasm adjuvants (baclofen, flupirtine, eperisone, tolperisone, thiocolchicoside) p.362 p.404 p.254
2.2.1	**Nausea and Vomiting 1**	• Physiology of N&V; emesis pathway; vomiting-centre physiology; receptors/ neurotransmitters; classification of anti-emetics (central/GIT) and prokinetics; pharmacological management of chemo-/radiotherapy-induced N&V p.87 p.276 p.393
2.2.2	**Nausea and Vomiting 2**	• Detailed pharmacology — metoclopramide, domperidone, 5-HT3 antagonists; anti-histaminic/anti-muscarinic drugs; psychotropics in N&V; miscellaneous (corticosteroids, benzodiazepines, NK1-receptor antagonists) p.87 p.276 p.248
2.2.3	**Constipation**	• Classification of aperients (laxatives); pharmacology of docusate, bisacodyl, lactulose, macrogol, senna, magnesium compounds, methylnaltrexone; rectal products (suppositories, enemas); opioid-induced constipation; constipation in paraplegia/ quadriplegia; drugs for diarrhoea p.383 p.281 p.403
2.3.1	**Cardiovascular drugs**	• Diuretics; optimising/stopping CVS drugs in the palliative phase; pharmacological management of cancer-associated thrombosis, DVT and PE p.393 p.43 p.64
2.3.2	**Respiratory drugs**	• Oxygen and intermittent/long-term oxygen therapy; bronchodilators (oral/parenteral/ inhaled); drugs for dyspnea, cough and respiratory secretions p.366 p.310 p.342

No.	Topic	Essentials
2.3.3	**CNS drugs (anxiolytics, antidepressants, antipsychotics)**	• Benzodiazepines — classification, pharmacology, rational use; prescribing antidepressants; drugs for delirium (typical/atypical antipsychotics); drugs for terminal restlessness (step-ladder and pharmacology of terminal sedation) p.402 p.248 p.335 p.477 p.491
2.4.1	**Topical Agents**	• Agents for dry mouth, sialorrhoea, mucositis, aphthous ulcers, oral candida; agents for dry skin, pruritus, pressure sores, malodorous/bleeding wounds; topical anal and eye preparations p.327 p.96 p.21 p.472
2.5.1	**Drug Interactions**	• Serotonin syndrome; QT prolongation; drug-induced movement disorders; synergistic sedation; metabolic (cytochrome P450) and pharmacokinetic interactions p.385 p.305 p.20 p.491
2.6.1	**Parenteral analgesic infusions**	• Preparing non-opioid/weak-opioid/strong-opioid infusions; syringe-driver preparation; compatibility and interactions; managing a patient on a syringe driver; drugs used in epidural/intrathecal analgesia p.382 p.296 p.402

2.7.1	**Prescribing in special situations**	•	Palliative drugs in renal dysfunction, hepatic dysfunction, cardiovascular morbidity, p.67 p.61 p.62
			children, the elderly, and cognitive impairment p.244 p.385 p.300
			p.15
			p.15
			p.15

SECTION CD3: SYMPTOM CONTROL IN PALLIATIVE MEDICINE

No.	Topic	Essentials
3.1.1	**Introduction to Pain** p.481	• Pain definition(s); pain taxonomy and classification(s); acute/chronic/cancer pain — approach and differences; breakthrough pain; pain crisis; Emory pain estimate model; general principles of managing pain in a palliative care setting p.360 p.271 p.250
3.1.2	**Mechanism of Pain 1**	• Anatomy of the pain pathway; peripheral and spinal pain mechanisms — nociception and anti-nociception; nociceptors; transduction, transmission, modulation and perception of nociceptive pain p.401 p.236 p.392
3.1.3	**Mechanism of Pain 2**	• Nerve injury; peripheral and central sensitisation; modulation in neuropathic pain; pathophysiological basis of hyperalgesia/allodynia; structural anatomy of bone in relation to malignant bone pain; pathophysiology of malignant bone pain p.373 p.236 p.392
3.1.4	**Assessment of Pain**	• Medical evaluation of a patient with pain; measurement and assessment tools (nociceptive and neuropathic); role of investigations/imaging; total-pain (psychological/psychosocial) evaluation; evaluating pain-associated impact and disability p.243 p.299 p.239
3.1.5	**Cancer Pain Syndromes**	• Acute cancer-related pain situations (diagnostic/therapeutic interventions, anti-cancer therapy, complications); chronic cancer-related pain situations (direct tumour-related, anti-cancer-therapy-related, paraneoplastic) p.29 p.238 p.330
3.1.6	**Cancer-Associated Nociceptive Pain**	• Visceral, genitourinary and vascular pain syndromes; cancer-related headache and facial pain; paraneoplastic nociceptive pain syndromes; lymphoedema-associated pain; inflammation/infection-associated pain p.238 p.69 p.106
3.1.7	**Malignant Bone Pain**	• Bone pain syndromes; pain in vertebral and long-bone metastasis; Mirels' scoring system; imaging modalities; management (analgesic step ladder, bisphosphonates, calcitonin, radiotherapy, radioisotopes, surgical interventions, chemo/hormonal/targeted therapy) p.373 p.294 p.238
3.1.8	**Cancer-Associated Neuropathic Pain**	• Direct nerve injury (plexopathies, painful mononeuropathy); paraneoplastic sensory neuropathy; malignant painful radiculopathy; painful cranial neuralgias; chemo-/RT-associated neuropathy; surgical neuropathies (phantom limb, post-mastectomy/thoracotomy); current neuropathic-pain-management guidelines p.332 p.363 p.293
3.2.1	**Nausea and Vomiting**	• Definitions and epidemiology; aetiological classification in palliative care; approach to a patient with N&V; opioid-, chemotherapy- and radiotherapy-induced N&V; aetiology-specific rational management p.276 p.87 p.99 p.489

No.	Topic	Essentials
3.2.2	**Constipation and Diarrhoea**	• Definition/classification; aetiology of constipation in palliative care; clinical approach and rectal examination; constipation assessment scales; management principles; opioid-induced constipation; constipation in paraplegia; assessment and management of diarrhoea p.383 p.281 p.299 p.489
3.3.1	**Dyspnea**	• Prevalence in life-limiting conditions; pathophysiology; physiological classification; assessment (quality, intensity, impact, distress); four-quadrant management approach (medical, rehab, palliative, EOL model); pharmacology of dyspnea; morphine and oxygen in dyspnea; non-pharmacological management; palliative sedation for intractable dyspnea p.305 p.393 p.43
3.3.2	**Cough, Haemoptysis, Respiratory Secretions, Bronchorrhoea**	• Cough pathway, causes, non-pharmacological and pharmacological treatment, refractory cough; haemoptysis classification, causes, assessment, management, interventions; respiratory secretions — prevalence, classification, presentation, management; bronchorrhoea p.366 p.310 p.326 p.489
3.4.1	**Delirium**	• Consciousness (awareness/alertness); neurophysiology; epidemiology and risk factors; clinical features; delirium-assessment tools; bedside assessment; hypoactive/ hyperactive/ mixed types; differential diagnosis; management (risk assessment, p.243 p.306 p.72
		prevention, safety, non-pharmacological and pharmacological treatment); terminal delirium p.335 p.274 p.397
3.5.1	**Miscellaneous symptoms 1 (hiccoughs, pruritus, sweats, dysphagia)**	• Hiccoughs — definition, pathway, aetiology, management, refractory hiccoughs; pruritus — classification, pathway, mediators, pharmacological and non-pharmacological management by type; sweats — aetiology, assessment, management p.393 p.43 p.299 p.489
3.5.2	**Miscellaneous symptoms 2 (fatigue and oedema)**	• Aetiology and pathophysiology of fatigue in a palliative care setting; clinical assessment and tools; non-pharmacological/pharmacological management; oedema — assessment and management (excluding lymphoedema) p.379 p.317 p.243

SECTION CD4: PALLIATIVE MEDICINE IN AN ONCOLOGY SETTING

No.	Topic	Essentials
4.1.1	**Cancer Epidemiology**	• Cancer trends in India (incidence and mortality); aetiology, risk factors, risk assessment (tobacco, infections, diet, lifestyle, physical/chemical factors); hereditary and familial cancer syndromes p.306 p.99 p.299
4.1.2	**Cancer Biology & Natural History**	• Cancer hallmarks (tumour biology, cell cycle, apoptosis, cancer stem cells, proto-oncogenes, tumour-suppressor genes, angiogenesis, invasion, metastasis); cancer genetics p.192 p.117
4.1.3	**Principles of Anti-cancer Therapy**	• Classification, pharmacokinetics/dynamics of anti-cancer drugs; indications, dose schedules, toxicity of commonly used drugs; hormone therapy p.29 p.351 p.126
4.1.4	**Palliative Surgery**	• Principles and indications in an oncology setting; common palliative surgical procedures (colostomy, ileostomy, gastrostomy, urinary diversion, tracheostomy, stenting, ERCP/PTBD and other interventional procedures); orthopaedic surgery in palliative care p.348 p.355 p.399
4.1.5	**Palliative Chemotherapy**	• Principles; definition and principles of adjuvant/neoadjuvant chemotherapy; indications and use of metronomic chemotherapy p.348 p.355 p.399
4.1.6	**Palliative Radiotherapy**	• Principles; role of RT in brain and malignant spinal cord compression, skeletal metastasis, visceral/soft-tissue metastasis; role of RT in haemostasis, analgesia and relief of obstructive symptoms p.91 p.277 p.336
4.2.1	**Head & Neck, Brain and Thoracic cancers**	• Stage-wise and palliative management of head & neck cancer; palliative RT and metronomic chemotherapy; management of low- and high-grade brain tumours; palliative RT for GBM with low KPS; palliative management of advanced oesophageal and lung cancer, dysphagia p.341 p.111 p.114
4.2.2	**Breast and Genito-urinary cancers**	• Stage-wise and palliative management of breast cancer; treatment algorithm for common GU cancers; palliative RT and chemotherapy for advanced GU cancers; palliation of obstructive uropathy p.82 p.90 p.129
4.2.3	**GIT Cancers including Hepatobiliary**	• Stage-wise management; palliative RT indications/schedules; palliative chemotherapy; palliation of bleeding, obstructive jaundice, malignant ascites p.365 p.309 p.351

No.	Topic	Essentials
4.2.4	**Paediatric cancers, soft-tissue tumours, leukaemia and lymphoma**	• Treatment algorithms for common paediatric cancers; palliative chemotherapy regimens for advanced/relapsed/recurrent disease; palliative RT indications and schedules p.21 p.356 p.351
4.3.1 / 4.3.2	**Neurological Complications & Emergencies**	• Malignant spinal cord compression (anatomy, epidemiology, clinical presentation, investigations, conservative management, RT/surgery, prognostication, evidence); status epilepticus; brain metastasis; raised intracranial pressure; encephalopathy (structural, metabolic, septic) p.91 p.290 p.93
4.3.3	**Haematological & Vascular Complications**	• Malignant SVC obstruction; DVT and PE; haemorrhage; tumour lysis syndrome; neutropenic sepsis p.286 p.15 p.47
4.3.4 / 4.3.5	**GI, Thoracic, GU, Bone and other Complications**	• Malignant bowel obstruction (pathophysiology, aetiology, proximal vs distal, investigation, conservative management, drugs, interventions, nutrition, prognostication); malignant ascites; malignant pleural/pericardial effusion; obstructive uropathy; pathological fractures; airway obstruction/stridor; managing pain crisis and opioid overdose p.81 p.365 p.309

SECTION CD5: PALLIATIVE MEDICINE IN A NON-ONCOLOGY SETTING

No.	Topic	Essentials
5.1.1	**End-stage Chronic Lung Disease (COPD)**	• Defining end-stage COPD; symptomatology; initiation of palliative medicine (Gold Standards Framework); 4-quadrant approach; dyspnea step-ladder; medical/rehab/ palliative models; opioids in dyspnea; guidelines for initiating EOLC; EOLC in end-stage COPD; palliative sedation in refractory dyspnea p.342 p.302 p.341
5.1.2	**End-stage Congestive Heart Failure**	• Defining end-stage CHF; illness trajectory models; heart-failure staging; symptomatology; initiating palliative medicine; referral triggers and guidelines; palliative approach; EOLC in CHF p.302 p.342 p.341 p.460
5.1.3	**CKD and End-Stage Renal Disease**	• Burden and symptom burden of ESRD; management of pain and non-pain symptoms; non-dialysis supportive care approach; managing EOL in patients on dialysis; guidelines for withholding/withdrawing dialysis p.341 p.301 p.395
5.1.4	**End-stage Liver Disease (ESLD)**	• Defining ESLD; symptom burden and management; EOL transitions (Child-Pugh/ MELD scoring); prognostication; palliative and EOLC approach in ESLD p.341 p.342 p.301
5.1.5 / 5.1.6	**Palliative Neurology — Symptoms, Impairment & Motor Neuron Disease** p.490	• Symptoms in advanced neurological illness (weakness, spasticity, dystonia, seizures, cramps, dyskinesia); impairment management (dysarthria, dysphagia, sialorrhoea, breathing difficulty, retention, bowel/bladder incontinence, sexual dysfunction, autonomic dysfunction); MND — classification, dysarthria, dysphagia/sialorrhoea, pain, dyspnea/NIV, interdisciplinary and end-of-life care p.289 p.288 p.39 p.462
5.1.7	**Other Neurological Conditions**	• Palliative care in cerebrovascular disease, demyelinating disease, Parkinson's disease, muscular dystrophy, Huntington's disease, traumatic/hypoxic brain injury, congenital and acquired peripheral neuropathy p.88 p.356 p.379
5.2.1 / 5.2.2	**Palliative Medicine in HIV/AIDS**	• HIV biology, natural history, phases; clinical course of AIDS; AIDS-defining complex; anti-retroviral therapy; infections in immunocompromise; non-infective complications; symptom prevalence; pain/non-pain symptom aetiopathogenesis and management; principles/approach, advance-care planning, stopping ART, infection prophylaxis; addressing non-physical issues p.395 p.407 p.354 p.464
5.3.1 / 5.3.2	**Palliative Medicine in Dementia**	• Epidemiology, pathophysiology and classification (Alzheimer's, frontotemporal, Lewy body, Parkinson's, Huntington's, vascular, HIV-associated); clinical features; DSM-5/ ICD-10 diagnostic criteria; psychiatric/neurological changes; course and prognosis; pharmacological/non-pharmacological treatment; palliative and end-of-life care in dementia p.343 p.354 p.397 p.466
5.4.1	**Haematological & Immunological disorders**	• Barriers to PC in incurable benign haematological disorders; sickle cell disease and thalassaemia major (inheritance, presentation, symptoms, needs, communication, long-term management); other congenital disorders; advanced vasculitis, malignant RA course, advanced SLE/PSS/MCTD/Sjögren's, progressive pulmonary fibrosis p.168 p.107

No.	Topic	Essentials
5.4.3 / 5.4.4	**Disability, MDR/ XDR-TB**	• Disability, impairment, activity limitation, participation restriction; rehabilitation–palliative care interphase; palliative care for traumatic paraplegia/quadriplegia, TBI/PVS, congenital disabilities; MDR/XDR-TB diagnostic criteria, clinical presentation, pharmacological management, EOLC needs, Geneva Declaration on Palliative Care and MDR/XDR-TB p.393 p.43 p.379 p.462

SECTION CD6: SUPPORTIVE CARE IN PALLIATIVE MEDICINE

No.	Topic	Essentials
6.1.1 – 6.1.3	**Managing Common Complications**	• Approach to shock (hypovolaemic diagnosis/management, types of shock, resuscitation fluids); fever and sepsis (definitions, bacteraemia/SIRS/sepsis/ septic shock/MODS, approach, complications, rational antibiotic use); anaemia in advanced illness (prevalence, approach, diagnostics, iron/ESAs, transfusion, symptom benefit, decisions on withholding transfusion) p.25 p.65 p.316
6.1.4 / 6.1.5	**Anorexia-Cachexia Syndrome & Thrombotic Disorders**	• ACS — definition/classification, aetiology, pathogenesis, staging, assessment, pharmacological management, nutrition; cancer-associated thrombosis; swollen legs in PC; recognition/confirmation/management of VTE; anticoagulation guidelines; SVC/portal/ cavernous venous thrombosis p.97 p.308 p.393
6.2.1 – 6.2.4	**Electrolyte and Acid-Base Disorders**	• Hyponatraemia/hypernatraemia (types, approach, treatment); hypo-/ hyperkalaemia; hypo-/hypercalcaemia and hypo-/hypermagnesaemia; general principles of acid-base balance; metabolic/respiratory acidosis and alkalosis p.54 p.52 p.53
6.2.5 – 6.2.8	**Infections in Palliative Medicine**	• UTI — definitions, risk factors, management, catheter-associated UTI, antimicrobial guidelines; respiratory infections (aspiration pneumonia, CAP, Pseudomonas, COPD exacerbation, viral/fungal); GI/hepatobiliary infections (diarrhoea, cholangitis, hepatitis, liver abscess, peritonitis, oral candida); skin/ soft-tissue/CNS infections (infected pressure sore/ wound, cellulitis, lymphangitis, herpes zoster, meningitis/meningoencephalitis) p.21 p.23 p.19
6.3.1 / 6.3.2	**Managing Co-morbid Illness**	• Diabetes mellitus in PC (prognosis-based glycaemic targets, EOL-phase management, insulin sliding scale, steroid-induced DM, DKA/HHS, hypoglycaemia); hypertension, IHD, dyslipidaemia, anti-platelets/anticoagulants, other co-morbidities (asthma, COPD, hypothyroidism, RA) p.56 p.55 p.342

SECTION CD7: PSYCHOSOCIAL ISSUES IN PALLIATIVE MEDICINE

No.	Topic	Essentials
7.1.1 – 7.1.3	**Illness Experience & Suffering**	• Human experience of illness; psychological response to illness; defining and understanding suffering; triangular model of suffering; dimensions of patient/ family distress; defense mechanisms and coping strategies; the emotional experience of pain, meaning of pain in terminal illness p.299 p.316 p.39 p.465 p.483
7.1.4 / 7.1.5	**Grief and Bereavement**	• Definitions (bereavement, grief, mourning, anticipatory/pathological/ disenfranchised grief); Kübler-Ross model; theories of grief; normal grief; bereavement-outcome factors; pathological grief, risk factors, prolonged grief disorder; bereavement follow-up and support; models of grief therapy p.353 p.306 p.400 p.469 p.486
7.2.1 – 7.2.7	**Psychiatry of Palliative Medicine**	• Distress and adjustment disorder (NCCN distress thermometer); depression in PC (screening, criteria, risk factors, treatment, suicide/desire for hastened death); anxiety subtypes; dealing with personality traits/disorders; severe mental illness in PC; neuropsychiatric changes with brain tumours/ leptomeningeal disease; the dying mind (twilight states, near-death experience, terminal restlessness) p.282 p.354 p.322 p.465 p.483
7.3.1	**Spiritual & Existential Issues**	• Defining spirituality, religion; spiritual distress and assessment tools; components of spiritual care; existential distress and its management p.375 p.307 p.354 p.485
7.4.1 / 7.4.2	**Psychosocial Support & Self Care**	• Types of caregivers; caregiver burden and measurement tools; interventions; support groups; burnout (definition, risk factors, markers); compassion fatigue; self-care assessment and protective practices p.376 p.377 p.315 p.484 p.492

SECTION CD8: PEDIATRIC & GERIATRIC PALLIATIVE MEDICINE, END OF LIFE CARE

No.	Topic	Essentials
8.1.1 – 8.1.8	**Pediatric Palliative Care**	• Children needing PPC (WHO Global Atlas); ACT/RCPCH trajectory groups I–IV; triaging; adult vs paediatric PC differences; pain assessment scales by age; WHO 2-step analgesic ladder for children; non-opioid/opioid dosing; paediatric delirium, dyspnea, cough, N&V, constipation; PPC in cancer and non-cancer conditions (neurodegenerative, haemolytic anaemia, cystic fibrosis, congenital heart disease, inborn errors); psychosocial/ethical issues, bereavement; adolescent palliative medicine p.300 p.355 p.408 p.469
8.2.1 – 8.2.3	**Geriatric Palliative Medicine**	• Sociodemographics and biology of ageing; frailty (definition, prevalence, pathophysiology, screening tools, falls risk); comprehensive geriatric assessment; common medical/psychological problems in the elderly; decision-making and goals of care p.429 p.354 p.243 p.468
8.3.1 – 8.3.4 p.473 p.474 p.484 p.488 p.492	**End of Life Care**	• Estimating EOLC need; gaps across settings; prognostication; principles/ components of Good Death; the 5 steps of providing good EOLC; recognising the dying process; ethics (autonomy, beneficence, non-maleficence, justice); futility and euthanasia; legal aspects; 6-step EOLC approach; respiratory secretions; palliative sedation; after-death care; death verification/certification; Registration of Births & Deaths Act 1969; IAPC/ISSCM joint-society 12-step EOLC guidelines p.348 p.390 p.366 p.468

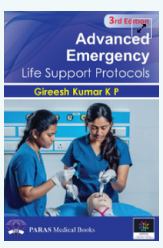
SECTION CD9: SPECIAL TOPICS IN PALLIATIVE MEDICINE

No.	Topic	Essentials
9.1.1 / 9.1.2	**Sleep, Body Image & Sexuality**	• Sleep physiology, theories, disturbances in advanced cancer, assessment tools, management; body image/sexuality across illnesses; psychosocial predictors of sexual functioning after cancer; PLISSIT model; interventions p.243 p.353 p.378
9.1.3 / 9.1.4	**Ethical Issues**	• Basics — cardinal principles (autonomy, beneficence, non-maleficence, justice), decision-making capacity/surrogate decision-making, confidentiality, informed consent; special situations — limitation of disease-modifying treatment, withholding/withdrawing life-sustaining treatment, nutrition and hydration, ethical situations in EOL decision-making, conflict/collusion, palliative care research p.304 p.348 p.343 p.481 p.488
9.1.5	**Advance Directives & Advance Care Planning**	• Definition, types, components and evidence for Advance Directives; definition, components and evidence for Advance Care Planning; differences between the two p.368 p.312 p.356
9.1.6 – 9.1.8	**Communication Skills Training**	• Basics and breaking bad news (patient-centred communication, active listening, SPIKES/ CLASS protocol, pitfalls, barriers); common communication issues (informed consent, decision-making, uncertainty, denial, collusion, conflict, anger, medical errors); advanced communication (cessation of disease-modifying care, transitions of care, discussing prognosis/future symptoms/goals of care/life-sustaining treatment/EOL communication) — all with role-play p.280 p.350 p.387 p.459 p.483
9.2.1 – 9.2.3	**Palliative Medicine in Special Situations**	• Bone marrow/stem-cell transplant (symptoms, psychosocial issues, pharmacology, communication, transitions/EOL); intensive care (indications, decision-making, communication, ethical/legal limitation of treatment); perinatal palliative medicine (scope, suitable conditions, fetal/neonatal pain assessment, antenatal/intrapartum/postpartum planning, EOL decisions with adverse prognosis) p.354 p.410 p.400
9.3.1 / 9.3.2	**Procedures & Interventional Techniques**	• Parenteral opioid infusions, syringe-driver set-up/compatibility/titration/ complications; epidural/intrathecal analgesia; site-specific neurolytic procedures; oxygen delivery/NIV/ tracheostomy; abdominal paracentesis, pleurocentesis, pericardiocentesis, intercostal drains; NG/NJ tubes, PEG/PEJ, ascitic/biliary drainage/stenting; urinary catheters including suprapubic/ percutaneous nephrostomy/DJ stenting p.405 p.382 p.296
9.4.1 / 9.4.2	**Integrative Medicine in Palliative Medicine**	• Classification; PC interphase; interventions; integrative medicine in pain, nausea, dyspnea, fatigue/anorexia-cachexia, anxiety/depression; evidence-based CPGs for integrative oncology and botanical preparations p.267 p.97 p.308

SECTION CD10: NURSING & REHABILITATIVE CARE IN PALLIATIVE MEDICINE

No.	Topic	Essentials
10.1.1 – 10.1.11	**Nursing Care**	• Stoma care — colostomy/ileostomy, tracheostomy, urostomy, gastrostomy (classification, management, complications, patient education); lymphoedema (anatomy, pathophysiology, staging, assessment, complete decongestive therapy); malignant wounds/fistulas/sinuses; pressure ulcers (risk scales, NPUAP staging, management, prevention); catheter care (CAUTI, insertion/ removal, types); oral care (mucositis, xerostomia, sialorrhoea, dysgeusia, oral candida, halitosis); urinary/faecal incontinence; nursing care of bedridden/altered-consciousness patients; nursing care at end of life p.96 p.70 p.391 p.470 p.472 p.490
10.2.1 – 10.2.4	**Rehabilitative Care**	• Quality of life, performance status and mobility (Karnofsky, ECOG, Barthel Index); rehabilitation team, needs assessment, goal setting, pulmonary/ speech-language/swallowing rehabilitation; rehabilitation for motor, sensory, cranial-nerve, cognitive deficits and deconditioning; nutrition and hydration in palliative medicine, enteral/parenteral nutrition in the terminally ill p.391 p.398 p.354

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3. Syllabus — Affective Domain (AD1–AD10)

Postgraduate trainees pursuing MD (Palliative Medicine) are expected to acquire the following attitudes and values. [AD = Affective Domain]

AD1: Palliative Care Principles

Code	Statement
AD1.1	Recognises pain, symptoms and suffering in patients with advanced life-limiting illness p.316 p.39 p.341
AD1.2	Recognises the need for relief of psychosocial, spiritual and existential suffering p.239 p.375 p.339
AD1.3	Recognises the need for appropriate care and support for the family and caregivers p.376 p.315 p.306
AD1.4	Recognises that care is person-centred, personalised and holistic, aiming to improve physical symptoms, suffering and quality of life p.377 p.266 p.389
AD1.5	Recognises the vast unmet palliative care needs in the population p.379 p.317 p.298
AD1.6	Understands principles of palliative care and its application p.348 p.399 p.389
AD1.7	Recognises the need to advocate for patients needing palliative care p.379 p.317 p.298
AD1.8	Understands various modes and models of palliative care delivery p.352 p.398 p.379
AD1.9	Recognises the need for palliative care policy at institutional/national level p.352 p.379 p.317
AD1.10	Recognises the need for palliative care quality standards and their implementation p.379 p.317 p.298

AD2: Pain & Symptom Management

Code	Statement
AD2.1	Demonstrates interest and openness in dealing with pain and symptoms p.282
AD2.2	Exhibits leadership and responsibility in dealing with poorly controlled pain and symptoms p.338
AD2.3	Exhibits safe prescription writing; recognises the need to identify aberrant drug use/diversion p.264 p.476
AD2.4	Recognises the role of cognitive, emotional and spiritual factors in the symptom experience p.267 p.375 p.354
AD2.5	Recognises the impact of pain and physical symptoms on ADLs, sleep, mood, sexual activity and social domains p.346 p.389 p.339
AD2.6	Recognises the value of a multidisciplinary approach to symptom management p.395 p.407 p.346
AD2.7	Recognises and initiates appropriate referral to other pain-management services p.300 p.393 p.340
AD2.8	Recognises the role of parenteral and interventional pain management in intractable pain p.372 p.314 p.300
AD2.9	Recognises the need to initiate palliative sedation in suitable patients with intractable symptoms p.305 p.351
AD2.10	Exhibits a compassionate attitude towards patients with pain and symptoms p.371 p.313

AD3: Clinical Expert

Code	Statement
AD3.1	Recognises palliative care needs in a patient with advanced cancer p.378 p.339 p.72
AD3.2	Advocates for early palliative care referral with the treating oncologist p.424 p.379 p.317
AD3.3	Recognises palliative care needs in non-oncology conditions (end-stage organ failure, advanced HIV/AIDS, chronic neurodegenerative conditions) p.263 p.341 p.302
AD3.4	Advocates for palliative care referral for advanced non-oncological conditions p.356 p.379 p.317
AD3.5	Recognises the importance of supportive care in length and quality of life p.321 p.369
AD3.6	Recognises complications and initiates management after considering benefits and futility p.101 p.89 p.104
AD3.7	Recognises co-morbid conditions and provides appropriate management or referral p.300
AD3.8	Recognises emergencies in palliative care p.347 p.406 p.379

Code	Statement
AD3.9	Recognises the importance of situation-specific care after considering benefits and futility p.375
AD3.10	Initiates appropriate referral that positively impacts symptom control and quality of life p.63 p.321 p.424

AD4: Psychosocial, Emotional & Spiritual Support

Code	Statement
AD4.1	Recognises the need for comprehensive assessment of socioeconomic, caregiver-support and living conditions p.376 p.345 p.315
AD4.2	Understands and evaluates psychological/emotional concerns of patients and families p.318 p.267 p.389
AD4.3	Recognises distress and exhibits an empathic approach p.375 p.278 p.271
AD4.4	Recognises the need for involvement of social workers/psychologists/counsellors as needed p.346
AD4.5	Recognises anxiety, depression and other psychiatric morbidity p.322 p.346
AD4.6	Recognises the need to consult psychiatric services when appropriate p.353
AD4.7	Exhibits a holistic approach towards psychiatric complications p.101 p.89 p.104
AD4.8	Recognises patients with self-harm behaviour and suicidal ideation p.371
AD4.9	Recognises spirituality as integral to a patient's experience p.307
AD4.10	Recognises that spiritual pain contributes to suffering, hopelessness and loss of meaning p.375 p.354 p.239

AD5: Multidisciplinary Care

Code	Statement
AD5.1 – AD5.10	Chooses to be a team player; recognises team cohesiveness; participates in and advocates during multidisciplinary/ interdisciplinary meetings (tumour board, joint clinics); respects opinions of team members; recognises the need for educational activity, research opportunities and team-building exercises within the team p.357 p.346 p.339

AD6: Shared Decision Making

Code	Statement
AD6.1 – AD6.10	Exhibits a non-judgemental attitude towards patient/family values and beliefs; participates in shared decision-making; recognises the importance of trust/respect/empathy; discusses therapies openly; recognises limitations and strengths of curative/disease-modifying treatment; participates in decisions on cessation of treatment, transitions of care, advance care planning, withholding/withdrawing life support and end-of-life decision-making p.304 p.348 p.312

AD7: Communication

Code	Statement
AD7.1 – AD7.10	Exhibits honest, accurate, sensitive information-sharing; recognises communication as essential to practice; exhibits effective/sensitive listening; times and discusses bad news appropriately; participates in discussion of emotional/ existential issues; exhibits competence in discussing transitions and EOL issues; exhibits willingness to discuss death and dying; exhibits leadership in complex communication; recognises confidentiality vs disclosure conflicts; values self-evaluation of communication skills p.350 p.280 p.400

AD8: Pediatric & Geriatric Care

Code	Statement
AD8.1 – AD8.10	Recognises varied presentation of pain/symptoms and needs of children across age groups; recognises the need for varied communication approaches with children, parents, grandparents and siblings; recognises how paediatric PC differs from adult PC; recognises multiple dimensions of old-age problems — frailty, disability, physical/psychosocial needs; recognises the importance of preserving function, preventing complications, managing comorbidity and maintaining dignity/QoL p.355 p.300 p.400

AD9: End of Life Care

Code	Statement
AD9.1 – AD9.10	Recognises the terminal phase; exhibits compassionate care of dying patients and families; exhibits readiness to continually care for the dying and support their family; exhibits a considerate, holistic EOL approach; recognises emotional challenges, grief and loss in self/staff/families; recognises EOL symptoms and initiates management; recognises non-physical needs and spirituality of the dying; recognises the importance of advanced, sensitive EOL communication; exhibits respect for the body after death, supporting religious/cultural practices; recognises the need for community awareness and institutional/national EOLC policy p.377 p.400 p.388

AD10: Professionalism & Altruism

Code	Statement
AD10.1 – AD10.10	Recognises own limitations and need for help/support; participates in personal reflection and mindful practice; acknowledges one's own potential issues of loss and grief; recognises care boundaries and manages expectations; respects colleagues' opinions while advocating for palliative care; exhibits leadership while respecting others within the interdisciplinary team; advocates for the socially disadvantaged/vulnerable; recognises the need to empower patients and families; recognises burnout in self and team, institutes early mitigation; recognises the importance of self-care and extending care to the team p.357 p.353 p.377

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4. Syllabus — Psychomotor Domain (PD1–PD10)

Postgraduate trainees pursuing MD (Palliative Medicine) are expected to develop the following procedural and non-procedural clinical skills. [PD = Psychomotor Domain]

PD1. Communication Skills

Establish rapport across ages, religions, cultures and socio-economic groups; obtain comprehensive history; comprehend information-sharing preferences; break bad news sensitively; handle denial, conflict and collusion; lead advanced communication (treatment cessation, transitions, goals of care); communicate clearly within the team; maintain accurate records.

PD2. Decision-Making Skills

Assess extent of desired patient/caregiver participation in decisions; facilitate participation in treatment decisions; discuss treatment options/continuation/cessation; conduct family meetings; lead decision-making on cessation of disease-modifying treatment, advance care planning, withholding/withdrawing life support and end-of-life decisions.

PD3. Pain & Symptom Management Skills p.479

Perform thorough history/examination and clinical assessment of pain and symptoms across age groups and settings; relate symptoms to pathophysiology; rationalise investigations; plan treatment per prognosis/ preferences; choose pharmacological treatment per age/renal/hepatic status; choose appropriate anti-cancer/ disease-modification for symptom control; handle/administer parenteral strong opioids; mix drugs in a syringe driver, know compatibilities, titrate doses; manage epidural/intrathecal catheters and assist simple neurolytic procedures.

PD4. Supportive Care & Disease Management Skills

Know natural history, epidemiology, anti-cancer therapies and transition points to facilitate early referral; estimate prognosis in advanced cancer; initiate referral for disease-modifying treatment or complication management; guide families on newer therapies; meet palliative care needs of end-stage organ failure, advanced HIV/AIDS, chronic neurodegenerative conditions; manage emergencies/complications (cord compression, SVC obstruction, airway obstruction, haemorrhage); manage concurrent illness and co-morbidities with appropriate specialist referral.

PD5. Psychosocial Support Skills

Assess and appraise psychological, social, financial, spiritual and existential concerns; identify and quantify distress; handle distressing emotions respectfully; identify spiritual issues/distress and provide spiritual care; perform mental-status examination, identify adjustment disorders/anxiety/depression; seek psychiatric help; identify self-harm/ suicide risk and initiate an emergency plan; discuss body image and sexuality sensitively; counsel patients and caregivers scientifically and rationally.

PD6. Multidisciplinary Care & Team Management Skills

Facilitate creation of a multidisciplinary team; work as a team member and team player; take up leadership; recognise contributions and delegate responsibilities; respect team members' opinions and resolve conflicts; attend and contribute to interdisciplinary meetings; carry out education, view-sharing and team-building; facilitate research opportunities.

PD7. End of Life Care Skills

Recognise the terminal phase and assist peers to do the same; participate in EOL decision-making with the team and with families; discuss preferred place of care; assess appropriateness and implement the EOLC pathway/ process; apply ethical and legal aspects of EOL care; assess and manage physical and non-physical needs of the dying; identify families at high bereavement risk; advocate for EOLC with peers, institution and community; advocate for institutional EOLC policy.

PD8. Procedural Skills p.479

Insert subcutaneous/intravenous lines and administer medication by these routes; set up a syringe driver, calculate doses, know compatibilities; handle syringe drivers, PCA pumps, continuous ambulatory devices; perform diagnostic/ therapeutic paracentesis and pleurocentesis; insert NG/NJ tubes and indwelling urinary catheters; recognise and manage pressure ulcers and malignant wounds (dressings, bleeding, malodour, myiasis); manage stoma care (tracheostomy, gastrostomy, colostomy) and perform high colonic enemas/ colostomy irrigation; use oxygen, nebulisers and non-invasive respiratory support; manage lymphoedema (complete decongestive therapy, bandaging, massage, exercise); plan and administer palliative sedation.

PD9. Quality Assurance, Education & Research Skills

Participate in departmental quality-assurance activity and audit; monitor programme effectiveness and reduce errors; develop institutional clinical algorithms/SOPs; teach and participate in educational programmes; conduct sensitisation programmes, CMEs and conferences; initiate/encourage research; seek IRB/ethics approval; declare conflicts of interest; conduct blinded randomised/observational studies; critically appraise RCTs and systematic reviews, exhibit evidence-based practice.

PD10. Good Practice & Leadership Skills

Identify own limitations and seek help; apply ethical principles day-to-day; uphold integrity, honesty and compassion; exhibit diligence, competence and approachability; apply mindful-practice principles; practise in an emotionally sustainable way; reflect on and understand personal loss and grief; detach individual values/beliefs when caring for patients with differing beliefs; work with mutual respect; care for self and the team.

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5. Teaching & Learning Methods

A. Formal teaching

Postgraduate trainees undergo formal teaching at departmental and institutional level. Teaching programmes are typically held on all working days, 8.30–9.30 AM (model schedule, adaptable by the institution):

Day	Duration	Activity
Monday	1 hour	Journal Club p.15 p.250
Tuesday	1 hour	Didactic Lecture p.15 p.250
Wednesday	1 hour	Subject Seminar p.15
Thursday	1 hour	Hospital Grand Rounds / Clinical meeting p.15
Friday	1 hour	Clinical Case Presentation p.15 p.43

- **Journal Club:** presentation of an original article (RCT/systematic review) or a short study with a review article — relevance, background, methods, statistical analysis, results, discussion, limitations and critical analysis. p.410
- **Didactic Lecture:** invited lectures on basic sciences, biostatistics, research/teaching methodology, medical ethics and legal issues, from external faculty, once a week. p.392 p.401
- **Subject Seminar:** a comprehensively prepared subject topic with literature search, covering clinical applications and audience engagement. p.239
- **Hospital Grand Rounds:** weekly attendance, presentations from various specialties related to Palliative Medicine. p.354 p.410
- **Clinical Case Presentation:** a case presented after thorough history/examination, eliciting physical and non-physical findings, formulating a diagnosis/differential and a comprehensive care plan. p.266 p.345

B. On-the-run (bedside) teaching

Trainees carry out clinical work under faculty/senior-registrar supervision — around 2 hours of dedicated teaching ward rounds each morning, plus on-the-run teaching in outpatients, consultation liaison, home care and hospice.

C. Additional teaching/training

Regular attendance at CMEs, conferences, workshops and small-group teaching organised locally/nationally/internationally, to stay abreast of current knowledge and recent advances.

D. Clinical postings — 3-year structure

Three years of supervised specialist training: 2 years Core Training in Palliative Medicine + 1 year Non-Core Training in related subjects (the non-core period does not exceed 1 year). Core-training clinical work spans ward and hospice, consultation liaison, community (home-based) care, and outpatients — admitting and assessing patients, managing pain/symptoms/complications/co-morbidity/emergencies, psychosocial and family-history taking, advance-care planning, multidisciplinary liaison, discharge planning, bereavement support, and day-care procedures (paracentesis, pleurocentesis, NG-tube insertion).

Year 1 (Core)	Year 2 (Non-Core)	Year 3 (Core)
Palliative Medicine – 12 months (OPD, ward, home-care postings)	3 months General Medicine + 3 months Medical Subspecialty (Gastroenterology, Neurology, Nephrology, Pulmonology, Cardiology, Endocrinology – 15 days each) + 1 month Paediatrics + 1 month Medical Oncology + 1 month Radiation Oncology + 15 days Surgical Oncology + 15 days Radiology + 15 days Public Health + 15 days Rehabilitation + 15 days Chronic Pain + 15 days Psychiatry	Palliative Medicine – 12 months (OPD, ward, home-care, consultation-liaison, hospice postings) p.354 p.410 p.389

6. Assessment Scheme

Formative assessment

Continual assessment of medical knowledge, patient care, procedural/academic skills, interpersonal skills, professionalism, self-directed learning and ability to practise in the system. Internal assessment should be frequent, cover all domains of learning, and used to provide feedback.

- Quarterly assessment based on: journal-based/recent-advances learning; patient-/laboratory-/skill-based learning; self-directed learning and teaching; departmental and inter-departmental learning activity; external/ outreach activities and CMEs – scored on the Postgraduate Student Appraisal Form (Annexure I). p.242
- **End-of-posting assessment:** supervisor scores personal attributes, work ethics, clinical work, interpersonal skills and communication; core-training trainees are evaluated quarterly, non-core trainees at the end of each posting. p.390 p.85
- **Academic-presentation assessment:** the moderator scores journal article, subject seminar and clinical-case presentations immediately after each presentation; a log book of work done must be maintained and periodically checked. p.85
- **Thesis-progress assessment:** every trainee must have a thesis guide, with progress checked and scored at 12, 18, 24 and 30 months. p.242

Summative assessment (as per Postgraduate Medical Education Regulations, 2000)

Two separate heads of passing – (A) Theory and (B) Practical/Clinical examination and viva-voce. Clearing one and failing the other amounts to failure of the summative examination.

Component	Structure & Passing Criteria
Theory examination	Four papers – **Paper 1:** Basic Sciences as applied to Palliative Medicine; **Paper 2:** Principles & Practice of Palliative Medicine (Cancer); **Paper 3:** Principles & Practice of Palliative Medicine (Non-Cancer); **Paper 4:** Recent Advances in Palliative Medicine. Cumulative pass ≥ 50%, minimum 40% in each paper. p.428 p.429 p.430
Clinical/Practical & oral examination	One long case (history, examination, interpretation of findings, differential diagnosis, investigations, prognosis, management) + three short cases (clinical management and communication/counselling skills), spread over two days if >5 candidates; may be conducted as an OSCE. Oral examination on drugs, instruments, radiological images, clinical images and charts. Pass ≥ 50%. p.242

RECOMMENDED READING

Core textbooks: Oxford Textbook of Palliative Medicine · Oxford Textbook of Palliative Medicine for Children · Oxford Textbook of Palliative Nursing. **Reference:** Palliative Medicine (Walsh) · Textbook of Palliative Medicine and Supportive Care · Evidence Based Practice of Palliative Medicine · The Psychiatry of Palliative Medicine · Palliative Care Formulary (PCF). **Journals:** 3–5 international and 2 national indexed journals.

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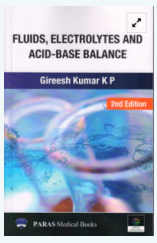
7. Recommended Reading & Postgraduate Appraisal Form (Annexure I)

Annexure I – reproduced from the NMC curriculum. A quarterly instrument scoring each trainee 1–9 (Not Satisfactory 1–3, Satisfactory 4–6, More than Satisfactory 7–9) across seven domains, with remarks and mandatory remediation suggested for any score below 4.

Sr. No.	Particulars
1	Journal-based / recent-advances learning p.198 p.170 p.141
2	Patient-based / laboratory- or skill-based learning p.370 p.286
3	Self-directed learning and teaching p.392
4	Departmental and inter-departmental learning activity p.248
5	External and outreach activities / CMEs p.373
6	Thesis / research work p.85 p.243
7	Log-book maintenance p.277

Plus: Publications (Yes/No); Remarks (any significant positive or negative attributes); signatures of assessee, consultant and HOD.

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SECTION 7

Essentials Supplement — IAPC & Twycross

Topics from the IAPC Textbook for the Certificate Course in Essentials of Palliative Care (5th ed., 2017) and Twycross's Introducing Palliative Care (4th ed.) that were missing or only briefly covered in Sections 1–6 — summarised in our own words, mapped to the NMC CBME competencies, and cross-linked to the main text. Topic 7.17 sets out every opioid conversion by the IAPC method, equation first.

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Blue page references (e.g. **p.250**) are live links back to Sections 1–6. Opioid conversions follow the IAPC method (7.17): breakthrough = 1/6 of the 24-h dose · oral : SC morphine = 2 : 1 · oral morphine ÷ 100 → fentanyl · rule of thumb ÷ 3.

7.1 Palliative Care — Definition, History, Principles & Models

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** Define palliative care. Enumerate the WHO principles of palliative care.
- **Q** Trace the history of the hospice and palliative care movement, with special reference to India.
- **Q** Describe the models of palliative care delivery. What is the Kerala model?

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe the history and philosophy of the hospice movement and of Indian palliative care (CD1.1.1)
- ✓ Define palliative care and state its principles (CD1.2.1, CD1.2.2)
- ✓ Describe levels of care and models of service delivery (CD1.3.1, CD1.9.1)

DEFINITION **WHO:** palliative care is an approach that improves the quality of life of patients and their families facing the problems associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems — physical, psychosocial and spiritual. The IAPC definition is given on p.432.

EXAM-ORIENTED THEORY

Milestones — world and India

Year	Milestone
273–232 BC	Emperor Asoka's institutions in India resembled hospices; a refuge for the dying at Varanasi
1842	The word "hospice" first used for a place for the dying (Lyon, France). *Hospes* = guest — root of hospice, hospital, hostel
1876	Hospice Desbassyns de Richemont, Pondicherry — the first hospice in India
1967	St Christopher's Hospice, London — Dame Cicely Saunders; birth of the modern hospice movement and the concept of "total pain"
1975 / 1984	India's National Cancer Control Programme included palliative care; in 1984 pain relief was made a basic service at primary health care level
1976	First hospital palliative support team — St Thomas' Hospital, London
1982	WHO Cancer Pain Relief Programme (Jan Stjernswärd) — took palliative care global
1986	Shanti Avedna Sadan, Mumbai — the first modern hospice in India (Dr L. J. de Souza)
1994 / 1996	IAPC registered as a national organisation; first international conference at Varanasi
April 2008	Kerala — the first Indian state to announce a palliative care policy
Later	MCI recognition of MD Palliative Medicine; a National Programme for Palliative Care drafted by MoHFW; NHM funds state palliative services

EXAM-ORIENTED THEORY

WHO principles of palliative care

- Relieves pain and other distressing symptoms
- Affirms life and regards dying as a normal process
- Intends neither to hasten nor to postpone death
- Integrates psychological and spiritual care
- Helps patients live as actively as possible until death
- Supports the family during the illness and in bereavement
- Uses a team approach, including bereavement counselling
- Enhances quality of life and may positively influence the course of illness
- Applies early in the illness, alongside life-prolonging therapy (chemotherapy, radiotherapy)

EXAM-ORIENTED THEORY

Types and models of palliative care

Type / model	Key features
Primary palliative care	Palliative approach by the first-contact doctor, nurse or health worker; identifies patients in the community; must be affordable and socially acceptable
Specialist palliative care	Interdisciplinary specialist team; works in three ways — direct care of complex patients, consultation to primary providers, education and support of EOLC services
Paediatric palliative care	Active total care of the child's body, mind and spirit, with family support
Hospital — consultation	Team advises the primary physician, who keeps primary responsibility
Hospital — OPD / inpatient	Out-patient clinic for mobile patients; designated beds or (better) a dedicated ward
Community / home-based	WHO public-health strategy; trained doctors, nurses, social workers and volunteers; Local Self Government Institutions sustain care (Kerala model — Neighbourhood Network)
Hospice	A philosophy of comfort care, not a building; only a few fully established hospices in India
Supportive care	From pre-diagnosis through treatment, cure or death, and into bereavement
End-of-life care	The last days, weeks or months; living as well as possible and dying with dignity in the preferred place
Rehabilitation	Faster response, quickly set realistic goals, constant adaptation to change

Building a team

- **Physical** — doctor and nurse (treating, prescribing); **psychological** — listening, counselling, being available; **social** — finance, housing, family support; **spiritual** — prayer, counselling, rites and rituals.
- Mutual respect, recognition of each member's work, shared load, training and supervision; volunteers are a major resource; reflect as a team after each death.

★ CLINICAL PEARLS & EXAM FOCUS

- Need in India (figures cited by IAPC, 2017): about 34 million people need palliative care and fewer than 1% have access; fewer than 3% of cancer patients get adequate pain relief.
- Exam favourite: first hospice in India = Pondicherry (1876); first modern hospice = Shanti Avedna Sadan, Mumbai (1986); first state policy = Kerala (2008).

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapters 1–2. Summarised and adapted in our own words for MD/DNB revision.

7.2 Communication — Collusion, Denial and Talking about Prognosis

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q What is collusion? How will you handle a family who says "Don't tell him it is cancer"?
- Q Denial as a coping mechanism — when should it be respected and when challenged?
- Q A patient asks, "Doctor, how long do I have?" How will you respond?

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Handle common communication issues — denial, collusion, conflict, uncertainty (CD9.1.6–9.1.8)
- ✓ Discuss prognosis honestly while maintaining realistic hope (CD9.1.6–9.1.8)

DEFINITION **Collusion** is the shielding of information about diagnosis or prognosis from the patient (usually by the family), or occasionally from the family by the patient. **Denial** is a coping mechanism by which a person avoids painful thoughts and feelings that are too difficult to face.

EXAM-ORIENTED THEORY

Handling collusion — a six-step approach

Step	What to do
1. Explore	Find out the family's main concerns and fears ("What worries you about him knowing?")
2. Acknowledge	Recognise that the request comes from love and a wish to protect
3. Explain benefits	Truth allows trust, shared decisions, completion of "unfinished business" and less isolation
4. Outline harms	Secrecy leads to mistrust, anxiety, isolation and missed chances to say goodbye
5. Challenge gently	If needed, challenge in a non-offensive way
6. Reassure	Promise that information will not be forced — the patient will be told only as much as he/she wishes to know

EXAM-ORIENTED THEORY

Denial

- Not wanting to know is a form of denial; it shows the patient is not yet ready to handle the news.
- **Respect** denial if the patient is co-operating with treatment and is not distressed — do not force information.
- **Gently challenge** it only when it prevents necessary treatment or causes distress (e.g. refusing care, unrealistic plans).

EXAM-ORIENTED THEORY

Talking about prognosis

- Avoid a direct number. First explore why the question is being asked (planning, fear, a family event).
- Speak in ranges — **days to weeks, weeks to months, months to years**. Over- or under-estimation causes great distress.
- Trend rule: deterioration **week by week** → likely weeks; **day by day** → likely days. A sudden fall over a few days — first look for a reversible cause (infection, hypercalcaemia, drugs).
- Useful phrase: "It is difficult to predict exactly; everyone is different. But he is clearly getting weaker."

EXAM-ORIENTED THEORY

Good communication technique

- Ask open questions; listen more than you speak
- Be empathetic; acknowledge emotions
- Balance hope and truth — never take away all hope
- Build a partnership between doctor, patient and family

★ CLINICAL PEARLS & EXAM FOCUS

- SPIKES for breaking bad news is covered on p.280 and p.387; this topic adds collusion, denial and prognosis, which SPIKES does not cover.
- A patient's understanding of what was said often differs from what the doctor thinks was said — check understanding at the end.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 3 (Communication skills) and chapter 24. Summarised and adapted in our own words for MD/DNB revision.

7.3 Palliative Care in End-Stage Heart Failure

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Compare the ACC/AHA staging and the NYHA functional classification of heart failure.
- Q Enumerate the similarities and differences between heart failure and malignancy from a palliative care perspective.
- Q Outline symptom management in end-stage heart failure. Which drugs should be avoided?

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Define end-stage heart failure, its trajectory and referral triggers (CD5.1.2)
- ✓ Manage symptoms and plan end-of-life care in heart failure (CD5.1.2)

DEFINITION **End-stage heart failure (ESHF)** is advanced structural heart disease with marked symptoms at rest or on minimal exertion despite maximal guideline-based treatment — ACC/AHA **stage D** and NYHA **class III–IV**. One-year mortality is about 50%.

EXAM-ORIENTED THEORY

ACC/AHA stage vs NYHA class

ACC/AHA stage	NYHA functional class
A — at risk, no structural disease, no symptoms	—
B — structural disease, no symptoms of HF	I — no symptoms
C — structural disease with prior/current HF symptoms	II — symptoms on moderate exertion · III — on minimal exertion
D — refractory HF needing specialised interventions	IV — symptoms at rest

Key facts

- Mainly a disease of old age (mean ~75 years); commonest causes — coronary disease and hypertension.
- About 40% die within a year of reaching end-stage disease; ~50% of deaths are sudden.
- Decline is slow and unpredictable, unlike the rapid terminal decline in cancer; prognostic models are unreliable.

Symptom burden

- Pain (angina, liver capsule stretch, arthritis), breathlessness, fatigue
- Anxiety and depression (a third of inpatients), anorexia, nausea, constipation, cachexia (poor sign), loss of libido
- Isolation from collusion and poor information

EXAM-ORIENTED THEORY

Heart failure vs cancer

Similarities	Differences in heart failure
Breathlessness, fatigue, pain	Life expectancy harder to predict; sudden death common
Weight loss, cachexia	Mistaken belief that it is more benign than cancer
Anxiety, depression, poor mobility	Oedema more prominent; no local pressure effects

EXAM-ORIENTED THEORY

Principles of symptom management

- **Optimise heart-failure treatment first** — ACE inhibitors and beta-blockers (improve symptoms and slow progression), diuretics titrated to fluid status (avoid over-diuresis: dizziness, nausea, fatigue).
- **Avoid drugs that worsen cardiac function** — NSAIDs and COX-2 inhibitors, tricyclic antidepressants, cyclizine.
- **Pain** — WHO ladder without NSAIDs; dose opioids as for renal impairment.
- **Nausea** — haloperidol for biochemical causes; metoclopramide for gastric stasis.
- **Anxiety / depression** — SSRIs (e.g. sertraline) or mirtazapine; safer than tricyclics for conduction and postural hypotension.
- **Breathlessness** — correct anaemia, low-dose opioid, non-drug measures from respiratory rehabilitation.
- **Non-drug measures** — salt and (in severe CHF) fluid restriction, nutrition, no smoking, limit alcohol, regular exercise, influenza and pneumococcal vaccines, adherence education.
- Help patient and carers live with uncertainty and a high chance of sudden death; plan advance care early.

★ CLINICAL PEARLS & EXAM FOCUS

- Breathlessness algorithm for end-stage COPD/heart failure: p.302; case: p.342.
- In patients with an implantable cardioverter-defibrillator, discuss deactivation of shocks as part of end-of-life planning (general practice point, not from the IAPC text).

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 10. Summarised and adapted in our own words for MD/DNB revision.

7.4 Palliative Neurology — MND, Multiple Sclerosis, Spinal Cord Injury, Stroke

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Discuss the palliative management of motor neurone disease.
- Q Autonomic dysreflexia — causes, clinical features and management.
- Q Outline the management of spasticity in a patient with multiple sclerosis.
- Q Palliative care issues in a patient with traumatic paraplegia.

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Manage symptoms and impairments in advanced neurological illness, including MND (CD5.1.5 / 5.1.6)
- ✓ Provide palliative care in traumatic paraplegia/quadriplegia and disability (CD5.4.3 / 5.4.4)

Most deaths in chronic neurological disease are due to complications (e.g. pneumonia) rather than the disease itself. Immobility and cognitive or behavioural change are more prominent than pain.

A

Motor neurone disease (MND / ALS)

DEFINITION Progressive degeneration of both upper and lower motor neurons causing wasting and weakness. Usually begins after 40 (mean onset ~58 years); about 40% survive 5 years. Riluzole is the only approved disease-modifying drug and prolongs life by only about 3 months.

- Spared: eye movements (III, IV, VI), bladder, bowel and sexual function; intellect, memory, sight and hearing are usually preserved.
- Bulbar involvement → dysarthria, dysphagia; UMN involvement → spasticity, emotional lability. **Dyspnoea is the most severe symptom.**
- Palliative care begins with breaking the news at the patient's pace, and includes counselling about costly alternative medicine, decisions on PEG and home ventilation, the fear of "choking to death", end-of-life care and bereavement.

Problem	Management
Weakness, cramps, fasciculations	Physiotherapy (not overdone), aids (cane, ankle–foot orthosis, wheelchair); cramps — magnesium, then quinine sulphate 200 mg bd or carbamazepine 200 mg bd; pyridostigmine 40–60 mg tds short trial (stop within a week if no benefit)
Pain (~73%)	Stiff joints — positioning, physio; inflammation — NSAID; joint pain — intra-articular steroid; spasm — diazepam/baclofen; skin pressure — repositioning, air/water bed
Insomnia	Usually from inability to turn, pain, fear of choking or dying; night sedation rarely causes respiratory depression in practice
Dysphagia, feeding	Semi-solid diet, early speech-therapy review; discuss PEG early — when choking or weight loss >10%, while vital capacity is >50% predicted; do not overfeed (aspiration); tube feeding may not prolong life at the end
Dysarthria	Early speech therapist; communication aids (light-writers, boards, computer devices) — plan ahead
Breathlessness	Chest physiotherapy; lorazepam 0.5–1 mg SL for anxiety; NIPPV for nocturnal hypoventilation (≥4 h/day, mainly at night), explaining its temporary nature and that stopping it will be supported with full comfort care; tracheostomy ventilation rarely used
Opioids	Oral morphine 2.5–5 mg titrated relieves breathlessness; used safely in >80% of patients in one study
Choking, laryngospasm	Choking is feared but rarely the cause of death — careful slow chewing, "breathe in, swallow, cough"; laryngospasm settles in seconds — repeated swallowing while breathing through the nose
Pathological laughing/crying (pseudobulbar affect, ~50%)	Ask specifically; amitriptyline 10–150 mg or fluvoxamine 100–200 mg

B

Multiple sclerosis (MS)

- Commonest inflammatory demyelinating CNS disease; usually **does not shorten life** — tell the patient. 70–80% start relapsing–remitting; half become secondary progressive within 10 years. Death is from complications (aspiration pneumonia, pulmonary embolism).
- "Depression is the most disabling, spasticity the most frequent, bladder symptoms the most confusing and bowel symptoms the most humiliating." Lifetime risk of depression ~50%; suicide risk 7.5× (highest in the first 5 years); cognitive impairment in 50–60%.
- **Pain** (~60%): neuropathic burning pain — TCA; trigeminal neuralgia — carbamazepine; Lhermitte's sign (electric shock on neck flexion) — collar ± carbamazepine; musculoskeletal and osteoporotic pain; headache.
- **Spasticity**: intensive physiotherapy and stretching, splints, TENS; treat triggers (UTI, pressure sores, constipation). **Baclofen** 5 mg tds, increase by 5 mg every 3–4 days to max 80 mg/day, withdraw slowly (fits, hallucinations); **diazepam** 2.5–5 mg tds or at night; **tizanidine** 2 mg, increase by 2 mg every 3–4 days to 24 mg/day (monitor LFTs for 4 months); gabapentin; nerve blocks (obturator, pudendal) or tenotomy if refractory.
- **Bladder**: hyperreflexic small bladder — oxybutynin; incomplete emptying (residual >100 ml) — clean intermittent self-catheterisation (fewer UTIs than indwelling); nocturia — desmopressin 10–40 µg nasal at night; indwelling catheter or diversion as last resort.
- **Fatigue**: avoid heat and hot baths, pace activities, rest periods, amantadine. Ataxia/tremor — occupational therapy, wrist weights; limited evidence for propranolol, clonazepam.

C

Spinal cord injury (SCI)

- Lesion above C4 → usually ventilator-dependent; cervical lesions affect arms, trunk and breathing; thoracolumbar lesions spare the arms.
- Kerala palliative data: prevalence >100 per million; commonest cause is **fall from height** (trees, construction sites, wells), then head-load injuries and road accidents; non-traumatic — transverse myelitis, tumours, spinal TB; men:women >5:1; mostly 20–40 years, poor manual labourers.
- **Neurogenic bowel**: UMN (above conus) — slow transit, hard stools, tight sphincter → softeners, fibre, fluids, suppository/digital stimulation; LMN — lax sphincter, incontinence + constipation → regular rectal emptying; train a bowel routine.
- **Skin** — insensate skin, low muscle mass, poor nutrition, wrong posture, spasticity → pressure sores (stage IV may need surgery). **Spasticity** — triggered by noxious stimuli (sores, bladder).
- **Pain** — neuropathic below the level (burning, electric), radicular, allodynia → TCAs, carbamazepine, gabapentin, valproate, opioids, baclofen, clonidine; shoulder overuse, compressive neuropathies, CRPS; visceral pain prevented by regular bowel and bladder emptying.
- **DVT** risk highest in the first 2 weeks and first year — prophylaxis; **bone** — osteoporosis, fractures, heterotopic ossification (calcium, vitamin D, bisphosphonates).
- **Sexuality** is a major unaddressed concern — counsel, encourage emotional bonding and other forms of intimacy. **Psychosocial** — loneliness, unemployment, loss of role → vocational rehabilitation.

Autonomic dysreflexia	
Mechanism	Uninhibited sympathetic discharge from a noxious stimulus below a lesion at **T6 or above**
Triggers	Bladder distension or UTI (check first), faecal impaction, pressure sore, ingrown toenail, fracture
Features	Sudden severe headache, paroxysmal hypertension (baseline in tetraplegia often <90/50, so 140/90 is high), brady- or tachycardia, flushing and sweating above the lesion, anxiety
Risks	Hypertensive crisis, seizures, arrhythmia, stroke
Management	Remove the stimulus (catheterise/unblock catheter, disimpact); sublingual nifedipine or topical nitroglycerine; prevent recurrences

D

Stroke (cerebrovascular accident)

- WHO: rapidly developing focal (or global) disturbance of cerebral function lasting >24 h or leading to death, with no apparent non-vascular cause. Infarction 84%, intracerebral haemorrhage 7%, SAH 7%.
- ¾ occur over 65 years, but in India ~1/5 occur below 40. ~20% recur within a year; more than a third die within a year and a third are permanently disabled. In cancer patients, stroke carries a median survival of about 4.5 months.
- Problems: altered consciousness (watch non-verbal distress cues), aphasia/dysarthria (~30%; assume the patient understands), cognitive deficits (~25%), delirium, seizures, DVT/PE, aspiration pneumonia, dysphagia, neurogenic bladder, pressure sores, spasticity, falls, deconditioning, depression (~1/3), central post-stroke pain (~2%) and hemiplegic shoulder pain.

- Rehabilitation from the acute phase into home care (physio, OT for feeding, dressing, bathing, one-handed techniques, splints); secondary prevention — BP, diabetes, lipids, smoking, antiplatelets or anticoagulation when embolic.

★ CLINICAL PEARLS & EXAM FOCUS

- MND: discuss PEG and ventilation **early** — the morbidity of PEG rises as vital capacity falls.
- Autonomic dysreflexia: think bladder first; sit the patient up and remove the trigger before giving drugs.
- MS is usually not life-shortening, yet suicide risk is high in the first 5 years — screen for depression.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 12. Summarised and adapted in our own words for MD/DNB revision.

7.5 Palliative Care in HIV/AIDS

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the role of palliative care in people living with HIV/AIDS in the ART era.

Q Enumerate the causes of pain in HIV/AIDS and outline its management.

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe the course of HIV/AIDS and the place of palliative care across it (CD5.2.1 / 5.2.2)
- ✓ Manage pain and non-pain symptoms and address non-physical issues in PLHA (CD5.2.1 / 5.2.2)

DEFINITION Palliative care in HIV (PEPFAR/OGAC) aims at optimal quality of life for people living with HIV/AIDS (PLHA) and their families through clinical, psychological, spiritual, supportive and preventive care **throughout the entire course of HIV infection** — not only terminal care.

Pre-ART era	Post-ART era
Rapidly fatal course	Chronic, manageable disease for many
Focus on opportunistic infections and palliative care	Unpredictable course, more prognostic uncertainty
Staff trained in end-of-life care	Complex regimens; focus of training shifted to ART, not palliative care

Why palliative care still matters

- ART is not a cure and has many side effects; co-infection with hepatitis B/C and malignancies can be fatal.
- Many symptoms at every stage; psychiatric illness and substance use; marginalised groups present late; adherence needs support.
- High social stigma — confidentiality is essential; often several infected (or dead) members in one family.

Components of care

Domain	Includes
Clinical	Prevention and treatment of TB and other opportunistic infections; symptom relief including pain; nutritional rehabilitation
Psychological	Counselling, support groups, treatment of depression, anxiety and HIV-related psychiatric illness, bereavement care
Spiritual	Culturally sensitive support through faith and rituals
Supportive	Child care, adherence support, legal help (rights, inheritance), housing, food, income generation
Prevention	Voluntary counselling and testing, support for sero-discordant couples, disclosure, condom use, mutual fidelity

Symptom management

- **Pain** — in >80% of hospitalised AIDS patients; 30–40% neuropathic. Causes: HIV itself (neuropathy, arthropathy, aseptic meningitis, infections, Kaposi sarcoma, lymphoma); treatment (procedures, ART neuropathy/myopathy, pancreatitis, radiotherapy); debility (pressure sores, constipation, wasting). Treat by the WHO ladder plus adjuvants.
- **Diarrhoea** — oral rehydration; if no blood, mucus or froth: loperamide 2 mg after each loose stool; if frothy, bloody or mucoid: metronidazole 400 mg tds for 14 days (IAPC regimen).
- **Fatigue/wasting** — may reverse with ART; late in the disease supplements rarely help — explain this.

- Treat specific conditions (candidiasis, TB, skin disease, bacterial pneumonia) when it improves quality of life without undue burden — even in advanced disease.

★ CLINICAL PEARLS & EXAM FOCUS

- India (NACO 2015 estimates quoted by IAPC): adult prevalence 0.26%, ~21.7 lakh PLHA, deaths falling with wider ART access.
- Advance care planning, decisions about stopping ART and infection prophylaxis at the end of life are listed in the NMC curriculum (p.438).

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 14. Summarised and adapted in our own words for MD/DNB revision.

7.6 Psychosocial Concerns — Fears, Anger, Losses, Depression & Suicide Risk

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Describe the psychological reactions of a patient with a life-limiting illness at different stages of the disease.

Q What are the risk factors for depression and suicide in a patient with advanced cancer?

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe the psychological response to illness, suffering, defences and coping (CD7.1.1–7.1.3)
- ✓ Screen for and manage depression, anxiety and suicidal ideation (CD7.2.1–7.2.7)

Phase of illness	Common reactions
Diagnosis	Shock, emotional numbness, disbelief, denial, anger
Treatment	Fear, sadness, guilt (may continue into the advanced stage)
Advanced / terminal	Regret, guilt, hopelessness, helplessness
All phases	Anxiety — at diagnosis, during treatment, at relapse and in the terminal phase

Fears

- Abandonment by carers or professionals
- Worsening pain and suffering
- More bad news
- Death, dying and the unknown
- Future of the family
- Treatment — procedures, side effects, outcome
- Unfinished business — children's education or marriage, property, forgiveness and reconciliation

Anger

- Delay in diagnosis; being sent from department to department
- Treatment failure after great expense
- Disfigurement (fungating wounds, lymphoedema, stomas, amputation)
- Uncontrolled pain
- Helplessness; blaming self, others or fate

Losses

- Composure and control
- Body function (colostomy, tracheostomy, resection)
- Appearance
- Role in family and society
- Dignity and respect
- Job and finances
- Relationships, intimacy and sexuality

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EXAM-ORIENTED THEORY

Depression

- "Depression" may mean existential distress, pessimism, reaction to stress, transient discouragement — or clinical depression. Look for persistent low mood ≥2 weeks, anhedonia, frequent crying, non-reactive mood, hopelessness, fearfulness; somatic features (fatigue, appetite, sleep) overlap with cancer.
- **Risk factors**: disability or disfigurement, social isolation, recent losses, uncontrolled pain or dyspnoea, past mood disorder or self-harm, advanced disease, substance use, negative life events, delirium.

EXAM-ORIENTED THEORY

Suicide risk — ask about it

Factor	Why it matters
Uncontrolled pain, multiple symptoms	Impulsive decisions when suffering is intolerable

Advanced illness, dependence	Frustration at loss of function; feeling a burden on the family
Helplessness / hopelessness	Especially after rejection by professionals or carers
Delirium	Confusion (metabolic, brain metastases, drugs such as opioids)
Finance, guilt	Unpayable treatment loans, job loss; guilt over past conduct, smoking, alcohol
Depression	Prolonged incurable illness, worry about family

Spiritual concerns

"Why me?", "Why is God letting me suffer?", "What is the point?", guilt, regret, a sense of being cursed, and beliefs about karma — which may also be a source of solace.

Helping patients cope

- Good pain and symptom control first — uncontrolled pain underlies many psychological problems, and untreated depression worsens pain.
- Explore and acknowledge concerns; counselling and education from diagnosis; psychotherapy; psychotropic drugs; family support.
- Maintain hope realistically, set realistic goals, schedule activities, and allow fears about dying to be spoken openly.
- Predictors of clinically significant distress: maladaptive personality or coping style, past psychiatric history, poor social or financial support, inadequate knowledge about the illness.

★ CLINICAL PEARLS & EXAM FOCUS

- Coping styles vary and may swing between fighting and acceptance — do not label normal reactions as pathological.
- Depression and anxiety algorithm: p.322.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 15. Summarised and adapted in our own words for MD/DNB revision.

7.7 Palliative Care in Dementia

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Discuss the diagnosis and assessment of dementia. Add a note on screening tools.
- Q Palliative care issues in advanced dementia. How will you counsel a family requesting a feeding tube?

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe the classification, DSM-5 criteria, course and treatment of dementia (CD5.3.1 / 5.3.2)
- ✓ Provide palliative and end-of-life care in dementia (CD5.3.1 / 5.3.2)

DEFINITION Dementia (DSM-5: **major neurocognitive disorder**) is a decline in cognition — learning and memory, language, executive function, complex attention, perceptual-motor or social cognition — severe enough to affect independence in daily life. About 20% of people over 85 are affected; the Dementia India Report (2010) estimated 3.7 million Indians with dementia.

EXAM-ORIENTED THEORY

DSM-5 criteria — major neurocognitive disorder

Criterion	Requirement
A	Significant decline from a previous level in one or more cognitive domains — concern of the person, an informant or clinician, **and** substantial impairment on standardised testing
B	Deficits interfere with independence (at least needing help with complex IADLs — bills, medicines)
C	Not occurring only during delirium
D	Not better explained by another mental disorder (depression, schizophrenia)
Severity	**Mild** — difficulty with IADLs; **moderate** — difficulty with basic ADLs (feeding, dressing); **severe** — fully dependent

Causes

- Alzheimer's, vascular, Lewy body and frontotemporal dementia — together 70–80%
- Others: Parkinson's, Huntington's, MS, normal-pressure hydrocephalus, brain tumour, subdural haematoma
- Reversible contributors: hypothyroidism, hypercalcaemia, hypoglycaemia, B12/thiamine/niacin deficiency, HIV, neurosyphilis, hepatic or renal failure, alcohol, anticholinergic drugs

Assessment

- **Cognition**: MMSE (serial change), Clock Drawing Test (executive function), Mini-Cog (clock + 3-word recall)
- **Function**: Functional Activities Questionnaire, ADLs
- **Behaviour**: BPSD in ~90% — hallucinations, delusions, agitation, aggression, wandering; **mood**: Geriatric Depression Scale
- **Physical**: examination, medication review, laboratory tests, CT/MRI where available
- **Caregiver**: burden, relationship, environment

EXAM-ORIENTED THEORY

Drug treatment (symptomatic only — may delay decline for 1–2 years)

Drug	Dose (IAPC)	Use
Donepezil	5 mg/day for 4 weeks, then 10 mg/day	Mild–moderate Alzheimer's (acetylcholinesterase inhibitor)
Rivastigmine	1.5 mg bd, up to 3 mg bd if tolerated	Mild–moderate Alzheimer's
Memantine	5 mg/day, up to 20 mg/day	Moderate–severe Alzheimer's or intolerance of AChE inhibitors (NMDA antagonist)

EXAM-ORIENTED THEORY

Palliative care issues

- **Pain** is under-recognised — shows as agitation or behaviour change; use observational tools such as the Abbey Pain Scale or DisDAT (see also p.313).
- **Antipsychotics for BPSD** only when symptoms are severe, dangerous or distressing, after non-drug measures; discuss risks; start low (risperidone 0.5–1 mg bd/HS, olanzapine 2.5–5 mg); stop if no response by 4 weeks; try to taper within 4 months; haloperidol not first line; avoid long-acting injectables.
- **Feeding problems** are characteristic of advanced dementia — treat reversible causes (teeth, constipation), modify texture, small portions, favourite foods; **careful hand-feeding is preferred to tube feeding**, which has not shown significant benefit — explain this with empathy.
- **Infections and fever** (respiratory, urinary) account for about a quarter of treatment decisions and are often terminal events — avoid indiscriminate antibiotics.
- **Advance care planning** early, while capacity remains — appoint a decision-maker; discuss preferred place of death (hospital transfers at the end of life are often avoidable and harmful).
- **Carers**: training, respite, 24-hour support, home-based care, bereavement support; a person with severe dementia is sometimes treated as socially "dead" — address this.

★ CLINICAL PEARLS & EXAM FOCUS

- Feeding decisions in advanced dementia: algorithm p.303, case p.343.
- Trajectory = prolonged dwindling; recognising the terminal phase and "dying from vs dying with dementia" are the key challenges.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 16. Summarised and adapted in our own words for MD/DNB revision.

Academy for Emergency Trauma & Critical care Medicine

7.8 Care of the Elderly — Geriatric Palliative Care & Policy

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q What are the common health, psychological and social problems of the elderly? How does palliative care help?
- Q How will you identify an older person approaching the end of life?
- Q Write a note on health care policies for older people in India.

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe the sociodemography and biology of ageing, frailty and comprehensive geriatric assessment (CD8.2.1–8.2.3)
- ✓ Recognise older persons approaching the end of life and plan care (CD8.2.1–8.2.3, CD8.3.1)

India's elderly population (~100 million) is projected to reach about 324 million — 20% of the population — by 2050. Many are poor, illiterate and live with multiple chronic diseases, frailty and dementia.

Problems of old age

- **Medical**: multimorbidity (diabetes, hypertension, IHD, cancer), sensory loss, arthritis, falls and fractures, incontinence, constipation, reduced cardiac/renal reserve
- **Psychological**: depression (commonest, often missed), anxiety, cognitive decline, dementia
- **Social**: financial insecurity (unorganised sector, no pension), loneliness, widowhood (mostly women), neglect and abuse, unawareness of government schemes
- **Spiritual**: multiple losses, fear about a good death, broken relationships, meaning and purpose

Special care issues

- **Polypharmacy** — interactions and adverse reactions; adjust doses for reduced liver and kidney function
- **Atypical presentation** — pneumonia without fever; delirium as the presenting sign of MI or even constipation
- **Geriatric syndromes** — multifactorial problems (falls, incontinence, delirium) outside single-organ categories
- **Prevention** — balanced diet, exercise, no tobacco or alcohol, social activity, fall-proofing the home (railings, walking aids, treating vision)

EXAM-ORIENTED THEORY

Identifying people approaching the end of life (GSF Proactive Identification Guidance)

Step	Action
1. Identify	The "surprise question" — would you be surprised if this patient died in the next year? If no, start end-of-life planning
2. Assess	Current and future clinical and personal needs
3. Plan	Living well and dying well — advance care planning, preferred place of care, carer support

"Approaching the end of life" (GMC/NICE) = likely to die within 12 months, including those whose death is imminent (hours to days). In the last 2–3 days, an integrated care plan for the dying person helps achieve a good death (see 7.12).

Care settings and support

- Home is the best place of care; short institutional stays for acute illness or respite; train the family, who are the main carers in India; old-age homes are increasing.
- National organisations: Indian Academy of Geriatrics, Alzheimer's and Related Disorders Society of India (ARDSI), HelpAge India — IAPC collaborates with them.

EXAM-ORIENTED THEORY

Policy for older people in India

Policy / law	Key points
National Policy on Older Persons (January 1999)	Financial security, health care, nutrition, shelter, education, welfare, protection of life and property; partnership with NGOs; active and productive ageing (IAPC)
Maintenance and Welfare of Parents and Senior Citizens Act, 2007	Legal duty of children/relatives to maintain parents and senior citizens; maintenance tribunals; old-age homes (added for completeness)
National Programme for Health Care of the Elderly (NPHCE), 2010	Dedicated geriatric services from sub-centre to district hospital and regional geriatric centres (added for completeness)

★ CLINICAL PEARLS & EXAM FOCUS

- Comprehensive geriatric assessment plus honest prognostication helps set treatment priorities for the frail elderly.
- Frail older people form the largest group needing palliative care.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 17; the 2007 Act and NPHCE rows are added from Government of India sources. Summarised and adapted in our own words for MD/DNB revision.

7.9 Children — Communication, Family Support and Bereaved Children

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q How will you communicate with a child who has a life-limiting illness and with the family?

Q Describe how children of different ages understand and respond to death. How can adults help a grieving child?

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Communicate with children, parents, grandparents and siblings; address bereavement (CD8.1.1–8.1.8)
- ✓ Describe grief and bereavement, including in children (CD7.1.4 / 7.1.5)

Communicating with the sick child

- Match information to the child's developmental level, understanding of illness and of death.
- Give clear, consistent, age-appropriate information — all professionals must say the same thing; stressed families may need it several times.
- Adolescents: counsel at the start; worries about appearance, vitality, dependency, fertility and sexuality; risk of non-adherence.
- Methods: talk, art, play, school work, drama, stories. **Children usually know more than their parents think** — encourage honesty.

Supporting the family

- Support from diagnosis through treatment and advanced disease; include siblings and grandparents.
- Inherited conditions — guilt and blame; offer genetic counselling and information on prenatal diagnosis.
- Open communication helps families cope best, though not every family works this way.
- Keep play and schooling going; short-term goals; inform and support teachers.
- After a child's death: support normal mourning, link to parent support groups, refer for specialist counselling if needed, withdraw contact gradually.

EXAM-ORIENTED THEORY

Bereaved children — reactions

- Denial (talks of the dead person as alive), guilt ("I caused it"), anger, anxiety that other carers will die, physical complaints, poor appetite, nightmares.
- Children grieve in spurts — "jumping in and out of puddles rather than wading through a river of grief".

Age	Understanding and response	How to help
Under 2–3 years	Little concept of death but aware of separation; protest, detachment, regression	Consistent carer, familiar routines, meet physical needs
3–5 years	Death not seen as permanent; worries about separation and abandonment; aggression, withdrawal, clinging, regression	Comfort, simple honest answers; include in rituals; keep mementoes; never describe death as "sleep" or "a long journey"
6–8 years	Seek causes; withdrawal, sadness, acting out, or becoming the "perfect child"	Short, honest, concrete explanations; normal activities and friends; allow brief regression; reassure they will be cared for
Pre-teen	Calmer, more accepting; good factual understanding	Encourage talking about the dead person; truthful answers; share your own feelings
Teenagers	Search for meaning and identity; withdrawal, depression, or anger and hostility; may hide fear behind jokes	Involvement, boundaries, take feelings seriously, keep peer contact, a trusted person to talk to

General approach — four principles

Principle	Practice
Information	Keep children informed; answer honestly in words they understand; it is fine to say "I don't know"
Involvement	Do not exclude them; explain in advance what will happen (viewing the body, funeral)
Reassurance	Extra comfort, familiar routines and boundaries; say what will stay the same and what will change
Expression of feelings	Show that feelings are acceptable — children learn this from the adults around them

★ CLINICAL PEARLS & EXAM FOCUS

- Paediatric pain and principles: p.300, p.340, p.355.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapters 18 and 26. Summarised and adapted in our own words for MD/DNB revision.

7.10 Ostomy and Tracheostomy Care

Section 7 contents ›

IAPC-based

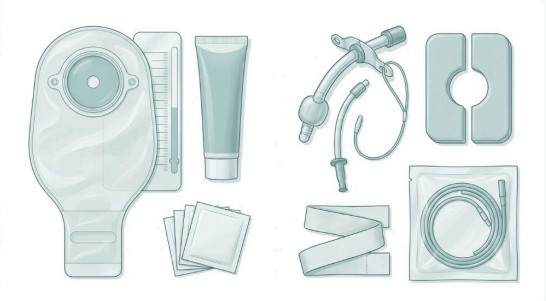
EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Classify stomas. Describe the care of a colostomy, including irrigation.
- Q Enumerate stoma and peristomal skin complications and their management.
- Q Describe the home care of a patient with a tracheostomy.

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Manage stoma care — colostomy, ileostomy, urostomy, tracheostomy, gastrostomy — and educate carers (CD10.1.1–10.1.11)

DEFINITION An **ostomy** is a surgically created opening for discharge of body wastes or for intake; the **stoma** (Greek, "mouth") is the visible opening. **Input** stomas: tracheostomy, gastrostomy, feeding jejunostomy. **Output** stomas: colostomy, ileostomy, urostomy (ileal conduit).



ILLUSTRATION

Stoma and tracheostomy care kit

- Two-piece appliance: cut the flange 2–3 mm larger than the stoma, using the measuring guide.
- Stoma paste or powder protects the peristomal skin; adhesive remover prevents stripping.
- Tracheostomy: clean the inner cannula regularly; change tapes with an assistant, one finger under the ties.
- Keep a spare tube, obturator and suction catheter at the bedside.

EXAM-ORIENTED THEORY

Colostomy

- Classified by duration (temporary/permanent), site (ascending, transverse, sigmoid) and construction (single-barrel, double-barrel, loop).
- Output depends on site — ascending (right): semi-formed; transverse (upper abdomen): semi-formed to formed; descending/sigmoid (left lower): formed.
- Pre-operative care: counsel patient and family, mark the site, explain what to expect; meeting another ostomate helps acceptance.

Stoma finding	Meaning / action
Colour	Normal deep pink to deep red — check regularly
Bleeding on rubbing	Surface capillaries — local pressure
Dusky or black	Ischaemia / necrosis — inform the surgeon immediately

Early oedema	Normal — no action
Herniation	Usually reduces lying flat; if it does not reduce, report urgently
Prolapse, retraction, stenosis	Usually need surgical correction

Peristomal skin

- **Mechanical** — frequent changes of adhesive bags → pectin-based powder ± sealant
- **Chemical** — leakage of stool or urine onto skin
- **Fungal** — moisture, sweating, antibiotics → antifungal powder
- **Allergic** — barrier or tape
- Clean with soap and water, pat dry; correct bag size; empty when **¾ full**; no creams or powders on intact skin; excoriation — karaya powder, or zinc oxide with oil; charcoal in the bag for odour

Living with a colostomy

- **Diet**: limit gas-forming and odour-producing foods (cabbage, onion, garlic, meat), fewer chillies; fibre and fluids against constipation; peppermint or chamomile tea for gas
- **Activity**: avoid rough games; protect the stoma when travelling and carry spare bags
- **Sexual life**: support, advice and counselling
- **Ileostomy**: liquid output; appliance always needed; never irrigate
- **Ileal conduit**: stoma and skin care, odour control, prevent UTI with plenty of fluids and citrus to keep urine dilute and acidic

Colostomy irrigation (IAPC)	
When to start	About 3 weeks after surgery; postpone (but start by 3 months) if on chemotherapy/radiotherapy or unwell
Volume	1–1.5 litres of water
Habit	Daily at a fixed time for the first 21 days, then continue lifelong
Do not	Irrigate during diarrhoea, or ever irrigate an ileostomy

EXAM-ORIENTED THEORY

Tracheostomy care

- Aims: a patent airway and removal of secretions. Classified as emergency or prophylactic, temporary or permanent, high (above thyroid isthmus) or low (3rd–4th tracheal rings). Tube parts: outer tube (tied in place), inner tube (locked), obturator (for insertion).
- Complications: accidental expulsion on coughing, wound and chest infection, choking from food or water entering the stoma.

Task	How
Inner tube	Remove and rinse under running water; soak in half-strength hydrogen peroxide, 2% sodium bicarbonate or saline; clean with soap and water; boil 3–5 min; reinsert and lock. Do **not** remove the outer tube
Skin	Clean around the stoma with saline gauze; slit gauze pad between flange and skin (petroleum gauze against excoriation); keep cleaning fluid out of the stoma
Suction	Only for copious, audible secretions; gentle, no longer than 5 seconds; prefer coughing and postural drainage
Humidification	Wet sterile gauze over the tube — humidifies and filters air
Tie	Change when soiled; one-finger gap between tie and neck
Speech and safety	Breathe in, occlude the tube with a finger and speak; bell or pen and paper; keep water out while bathing or shaving; keep insects out; oral hygiene; teach self-care with a mirror

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 19. Summarised and adapted in our own words for MD/DNB revision.

7.11 Nursing Care of the Bedridden Patient

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Describe the components of total nursing care of a bedridden or unconscious patient.
- Q Pressure sores — staging, causes and prevention.
- Q How will you manage maggots in a fungating wound?

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Provide nursing care of bedridden and altered-consciousness patients; catheter, oral and pressure-area care (CD10.1.1–10.1.11)
- ✓ Use topical agents for mouth, skin and malodorous wounds (CD2.4.1)

Major problems in a bedridden patient: corneal ulcer, pressure sores, malnutrition, psychological distress and electrolyte imbalance. **Total care** = personal hygiene, nutrition, active and passive exercise, bladder care, bowel care, communication and symptom treatment. The unconscious patient also needs airway care, fluids (NG or IV) and protection of the eyes and skin.

Hygiene

- **Mouth care** — saline, soda bicarbonate, lime juice or water boiled with neem leaves; teach carers for unconscious patients
- **Bed bath** — privacy, warm draught-free room, expose one area at a time, clean to less-clean, soak hands and feet, trim nails, dry creases and folds, inspect the back for early sores, moisturise
- **Back care** — at least 2-hourly for those at risk; wash, pat dry, massage pressure points with lubricant
- **Hair** — head bath on a mackintosh into a bucket; brushing and massage aid circulation
- **Eyes** — inner to outer canthus, separate swab for each eye; **nose and ears** — moist applicator; wax softened with oil
- **Perineum** — clean from urethra (cleanest) towards anus; after every void or bowel motion

Pressure sores

Stage	Depth
1	Skin intact but inflamed
2	Broken into epidermis/dermis
3	Into subcutaneous tissue
4	Into muscle or bone

- **Causes**: pressure (>2–3 h), friction, moisture, infection; predisposed by poor circulation, oedema, emaciation, obesity, paralysis, sedation, incontinence, diabetes, fever
- **Prevention**: identify at-risk patients; inspect daily; turn **2-hourly lying, 1-hourly sitting**; air or water bed, cushions; clean, dry, lubricated skin; protein-rich diet and fluids; wrinkle-free sheets; lift (do not drag) on and off bedpans

EXAM-ORIENTED THEORY

Bladder and bowel

Area	IAPC practice
Urinary catheter	Correct size; balloon 5–10 ml sterile water; clean daily; change latex Foley every 3–4 weeks, silicone every 3 months; fluids 2–3 L/day; bag below waist level, cap closed
Opioid patients	Start a softener plus a stimulant (docusate with senna or bisacodyl) together with the opioid
No motion for 2 days	Increase laxatives
No motion for 3 days	Rectal examination — soft stool: bisacodyl 10–20 mg or suppository; hard stool: manual evacuation then glycerine or phosphate enema
Partial bowel obstruction	Softener/osmotic only; avoid stimulant laxatives
Spinal cord compression / paraplegia	Keep stool firm — stimulant (senna) without softener; regular manual evacuation
Diarrhoea on laxatives	Stop for 24 h, then restart at a lower dose

EXAM-ORIENTED THEORY

Wound and mouth extras

Problem	Management
Maggots (myiasis) in a wound	Flush plain turpentine into the wound with a syringe, wait 10 minutes, remove maggots with forceps; repeat turpentine dressings for 3–4 days (IAPC practice)

Wound infection / malodour	Wash under running water or saline; systemic antibiotics if spreading cellulitis; topical metronidazole gel or crushed tablets in lignocaine gel for smell (see p.318, p.380)
Halitosis	Causes: poor oral hygiene, caries, gingivitis, necrotic tumour, sinus or suppurative lung disease → frequent mouthwash, metronidazole gargle or course, antibiotics for infection
Altered taste	Hypogeusia/ageusia or dysgeusia (metallic taste) → mouth care, treat candida, adjust diet; pickles, lemon, salt or vinegar to stimulate taste; more fruit and water; food temperature as preferred

★ CLINICAL PEARLS & EXAM FOCUS

- A homemade soft "toothbrush" (gauze-wrapped handle) cleans a sore mouth with less pain and bleeding.
- Aim of laxatives = comfortable defecation, not frequency; 40–60% of terminally ill patients still need rectal measures.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapters 7, 20 and 22. Summarised and adapted in our own words for MD/DNB revision.

7.12 The Good Death — Ten Principles of Care for the Dying Person

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What is a "good death"? Enumerate the principles of a good death.

Q List the ten principles of best care for the dying person. Why should the team diagnose dying?

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Describe the principles and components of a good death and recognise the dying process (CD8.3.1–8.3.4)

✓ Plan care in the last days of life and after death (CD8.3.1–8.3.4)

DEFINITION A **good death** is free from avoidable distress and suffering for patients, families and carers, in general accord with their wishes, and reasonably consistent with clinical, cultural and ethical standards. In 2014 the IAPC position statement declared a good, peaceful and dignified death a human right.

Principles of a good death

- Know when death is coming and what to expect
- Retain control over what happens
- Dignity and privacy
- Control over pain and other symptoms
- Choice of place of death
- Information and expertise as needed
- Spiritual and emotional support
- Hospice care in any setting
- Control over who is present at the end
- Advance directives respected
- Time to say goodbye
- Not having life prolonged pointlessly

EXAM-ORIENTED THEORY

Ten principles of best care for the dying person (BMJ 2013)

No.	Principle
1	Recognition by the MDT (at least doctor and nurse) that the person is in the last hours or days, documented by the senior doctor
2	Communicate this recognition to the person, where appropriate, and to those important to them
3	Opportunity to discuss wishes, concerns, feelings, faith, beliefs and values
4	Anticipatory prescribing for expected symptoms
5	Review every clinical intervention in the person's best interest
6	Review hydration — start, continue or stop clinically assisted hydration
7	Review nutrition — continue or stop clinically assisted nutrition
8	Full discussion of the care plan with the person and family
9	Regular reassessment (IAPC: at least 4–6-hourly) and MDT review (24–48-hourly)

10 Dignified, respectful care of the person and family immediately after death

EXAM-ORIENTED THEORY

Why diagnose dying?

- Prevents futile investigations and interventions, unrealistic expectations, unwanted ICU or hospital transfers, uncontrolled symptoms, loss of trust and dissatisfaction, and difficult bereavement.
- Allows the family to prepare, finish legal and bank matters, gather relatives for goodbyes, and plan rites (e.g. Ganga jal, tulsi, a priest).

Care after death

- Care of the body with dignity and attention to cultural and religious practice; focus moves to the family.
- Review the process — debrief, support staff, audit. Practical steps (certification, registration, donation) are in 7.13.

★ CLINICAL PEARLS & EXAM FOCUS

- Last hours of life: algorithm p.275; death rattle p.310; terminal delirium p.274; planning death at home p.329.
- Most dying patients are not looked after by palliative specialists — every clinician needs these skills.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 24. Summarised and adapted in our own words for MD/DNB revision.

7.13 Practical Issues after Death — Certification, Registration, Donation

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q A patient dies at home. Advise the family about the death certificate, keeping the body, organ donation and the legal formalities.
- Q Write a note on the Registration of Births and Deaths Act, 1969.

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe death verification and certification and the Registration of Births and Deaths Act, 1969 (CD8.3.1–8.3.4)

Issue	What the family and doctor need to know
Care of the body at home	Clean and dry the body, close the eyes and mouth. If relatives must travel, use embalming, a mortuary or a portable freezer — there is no need to rush the funeral
Medical Certificate of Cause of Death (MCCD)	Issued by the hospital or hospice when death occurs there. For a death at home, contact the ASHA worker, who involves the local doctor to certify death; a certificate is also needed to transport the body
Registration (RBD Act 1969)	Every death must be registered with the State/UT within **21 days**; the head of family or ASHA informs the municipality or panchayat. Late registration needs the Registrar's or Magistrate's permission and a fee
Death certificate	Legal document stating date, fact and cause of death — needed for inheritance, insurance, family pension and closing obligations
Organ and tissue donation	Transplantation of Human Organs Act 1994, amended 2011, rules 2014. Tissues such as cornea, sclera, heart valves, bone, tendons and skin can be donated after death; if the person had a donor card, the next of kin must contact the designated person in time
Post-mortem	Medico-legal post-mortem only if unnatural death or uncertain cause — may proceed without consent; a hospital (pathological) autopsy needs family permission
Funeral	Honour the person's wishes (e.g. cremation); funeral service providers exist in some cities; memorial service or trust
Legal and financial	Execute the will; without a will apply to the Tahsildar for a legal-heir certificate; notify employer, pension office, banks, insurers, utilities, licence and passport authorities, and the family doctor

★ CLINICAL PEARLS & EXAM FOCUS

- Explain to the family that grief — disbelief, longing, anger, guilt, numbness — is normal even when death was expected.
- NMC lists death verification, certification and the RBD Act 1969 in CD8.3 (p.440).

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 25. Summarised and adapted in our own words for MD/DNB revision.

7.14 Morphine Availability — NDPS Act 2014, ENDs and RMIs

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q What were the reasons for poor availability of opioids in India? How did the NDPS Amendment 2014 change this?

Q Define Essential Narcotic Drugs and Recognised Medical Institution. Describe the steps to obtain RMI status.

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe the NDPS Act and its amendments, and national palliative care policy (CD1.9.1)
- ✓ Advocate for availability of essential pain-relief drugs (CD1.8.1)

DEFINITION Opioids are controlled under the **Narcotic Drugs and Psychotropic Substances (NDPS) Act, 1985** (Department of Revenue, Ministry of Finance). The **NDPS Amendment Act, 2014** and **NDPS Rules, 2015** introduced the principle of **balance** — preventing diversion while ensuring adequate availability for medical use.

Why opioids were unavailable

- Prohibitory colonial-era laws; each state had different licences and quotas
- Multiple licences from several agencies; severe penalties (even for clerical errors) alienated pharmacists
- Professionals trained only with injectables; exaggerated fear of addiction and respiratory depression ("opiophobia")
- Public belief that morphine means addiction or impending death

What the 2014 amendment did

- Notified a short list of **Essential Narcotic Drugs (ENDs)** — morphine, fentanyl, methadone, oxycodone, codeine, hydrocodone
- Uniform national rules for ENDs instead of state rules
- One state agency — the **State Drugs Controller** — recognises institutions as RMIs for 3 years
- No more multiple licences from multiple agencies

Term	Definition (NDPS Rules 2015)
Essential Narcotic Drug	NDPS-controlled medicine on the notified list, for moderate to severe pain, used in an RMI
Registered Medical Practitioner	Registered doctor or dentist trained in the medical use of ENDs
Recognised Medical Institution (RMI)	Medical institution recognised by the State Drugs Controller to purchase, possess and dispense ENDs. Needs: a trained doctor in charge, a double-lock cupboard, infrastructure and staff, and record-keeping (Forms 3D, 3E). Government hospitals are deemed RMIs if they meet these

EXAM-ORIENTED THEORY

Getting and keeping RMI status

Step	Process
1. Training	A registered doctor is trained in the medical use of opioids
2. Apply	Form 3F to the State Drugs Controller, with the doctor's degree, registration and training certificates, Form 3J (annual estimate of ENDs and source), covering letter
3. Inspection	Recognition letter (Form 3G) — or reasons for refusal — within 60 days; states the annual quantity
4. Purchase	Order to the supplier with a copy of the RMI certificate
5. Receipt	Consignment arrives with Form 3C — keep the original, return the copy

6. Records	Daily stock register (Form 3D) and patient-wise register (Form 3E), kept for 2 years; annual return (Form 3I) by 31 March; doctor in charge signs the daily balance
Renewal	Before 30 November each year — balance, purchases, quantity dispensed and next year's need; revise the annual estimate by 31 August if needed; keep at least 3 months' buffer stock

Small stock without an RMI (emergency use by any RMP)

Morphine ≤500 mg; codeine ≤2000 mg; hydrocodone ≤320 mg; oxycodone ≤250 mg; fentanyl — two patches (12.5 and 25 µg/h). More needs special permission (Form 3B, valid 3 years).

Prescribing an END

In capital letters, dated and signed, with the doctor's full name, address and registration number; patient's name and address; total quantity, daily dose and duration.

★ CLINICAL PEARLS & EXAM FOCUS

- The rules were under review when the IAPC text was written — check the Department of Revenue website for current forms and limits.
- Policy context: p.352; substance use and stewardship: p.264.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 27. Summarised and adapted in our own words for MD/DNB revision.

7.15 Safe Use of Opioids — Chronic Non-Cancer Pain, Myths and Aberrant Use

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Discuss the principles of long-term opioid therapy in chronic non-cancer pain.
- Q What are the myths about morphine? How will you detect aberrant drug-taking behaviour?

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Describe adverse effects of long-term opioid use and safe prescribing (CD2.1.2, CD2.1.3)
- ✓ Recognise aberrant drug use and diversion (AD2.3–2.4)

Ground rules for long-term opioids

- Goal = better function and quality of life, not just a pain score
- Never prescribe strong opioids where follow-up is impossible
- Discuss risks, realistic benefits and goals before starting
- Keep non-opioid and non-drug treatments; address psychosocial contributors
- Continue only while benefit clearly outweighs risk

Selecting the patient

- Is the pain opioid-sensitive?
- Major psychological contributors? Negative thoughts that can be addressed?
- Is there a non-drug option?
- Can progress be followed up?
- Cancer pain: consider opioids early. Non-cancer pain (therapy for decades): only after non-drug and non-opioid options fail

EXAM-ORIENTED THEORY

Titration and review (non-cancer pain)

- Always start with **immediate-release**, never extended-release, opioids.
- Reassess carefully at **50 mg** oral morphine/day; justify any increase to **90 mg**/day.
- Review benefit and harm within 1–4 weeks, then every 3 months; if harm outweighs benefit, taper and stop.
- Monitor the **4 As**: Analgesia, Activity, Adverse effects, Aberrant behaviour. Policy: "trust but verify".

Myth (among professionals)	Fact
Physical dependence happens only with high doses for very long periods	With daily use, dependence can develop within days or weeks
Opioids can easily be tapered and stopped	Tapering after long use can be difficult, with prolonged, distressing withdrawal
Overdose happens only in drug abusers and suicide attempts	Long-term users risk unintentional overdose, more with higher doses, extended-release forms, benzodiazepines and alcohol

Oral morphine for cancer pain causes addiction	Given for the right pain in the right dose it rarely does; injectable misuse produced the "kick", not oral morphine

Long-term adverse effects

- Chronic constipation and faecal impaction
- Dry mouth → dental decay
- Unintentional respiratory depression
- Falls from dizziness
- Hypogonadism
- Opioid-induced hyperalgesia
- Sleep-disordered breathing

Aberrant behaviours

- Focus on getting opioids; refuses alternatives; asks for a particular drug; upset when refused
- Repeated ER visits for pain; supplies used up early; reports of lost or stolen medicine
- Other substance use (alcohol, benzodiazepines)
- Poor function with unclear cause of pain that flares with minor stimuli
- Higher risk: past or family history of substance use, adolescents/young adults, depression

Adherence monitoring feasible in India

One prescriber and one pharmacy; pill counts (empty strips at each visit); check reports of lost medicine. Do not abandon a patient with a drug problem — offer help or refer.

Opioids for acute pain — best practice

Short courses only; immediate-release for the day and next day; avoid SR and transdermal forms; cover side effects; review; step down early to non-opioids.

★ CLINICAL PEARLS & EXAM FOCUS

- Kozhikode experience (Lancet, 2001): 1723 home-care patients on oral morphine over 2 years — no misuse or diversion identified, with careful selection, follow-up and record keeping.
- Substance use and opioid stewardship: p.264.

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapters 6 and 28. Summarised and adapted in our own words for MD/DNB revision.

7.16 Quick Formulary — Commonly Used Drugs (IAPC)

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q List the commonly used drugs in palliative care with their doses and cautions.
- Q Give the equivalent doses of commonly used corticosteroids.

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Prescribe common palliative drugs safely, including adjuvants and CNS drugs (CD2.1.4, CD2.3.3)

Drug	Indication	Dose · route · frequency	Caution / interaction
Paracetamol	Mild–moderate pain, fever	0.5–1 g PO 6-hourly (max 4 g/day); child 10–15 mg/kg; IV 1 g over 30 min	Hepatic and renal impairment
Ibuprofen	Mild–moderate pain	200–400 mg PO tds	Peptic ulcer, GI bleed, renal risk (myeloma, hypovolaemia); bleeding with anticoagulants or steroids
Diclofenac	Mild–moderate pain	50 mg PO tds; 75 mg IM bd; 100 mg PR daily	As for NSAIDs
Tramadol	Moderate pain	50–100 mg PO 4–6-hourly, max 400 mg/day	Epilepsy, raised ICP; serotonergic drugs; not with MAOIs
Morphine IR	Severe pain	5–10 mg PO 4-hourly, double dose at bedtime; increase 30–50% as needed	Renal failure; always with laxative
Morphine SR	Stable pain	24-h IR total ÷ 2, 12-hourly	Never start with SR

Morphine SC	Unable to swallow	Half the 24-h oral dose over 24 h (CSCI)	See 7.17
Morphine (dyspnoea, cough)	Breathlessness, cough	2.5–5 mg PO 4-hourly and PRN	—
Fentanyl patch	Stable severe pain	25 µg/h start (opioid-tolerant), 72-hourly; convert as in 7.17	Fever or heat raises absorption
Amitriptyline	Neuropathic pain	10–25 mg HS (10 mg elderly), increase every 3–4 days to 75 mg/day	Glaucoma, retention, arrhythmia, recent MI
Gabapentin	Neuropathic / bone pain	100–300 mg OD → BD → TDS over 6 days; up to 1200 mg tds	Elderly, renal failure
Pregabalin	Neuropathic pain, anxiety	25 mg OD → BD → 75 mg BD over 6 days	Elderly, renal failure
Dexamethasone	Bone/nerve pain, dyspnoea	4–8 mg PO mornings	Hyperglycaemia, myopathy, mood
	Nausea; bowel obstruction	12–16 mg (N&V); 8–16 mg (obstruction, SVCO, cord compression)	
	Cerebral oedema	8–16 mg stat, then 4 mg 6-hourly IV; up to 16 mg CSCI	
Ondansetron	Chemo/RT nausea; pruritus	4–8 mg PO/SC/IV bd–tds	Bowel obstruction, QT
Granisetron	Nausea	1 mg stat then bd	—
Haloperidol	Toxic/drug nausea, delirium, agitation	1.5–3 mg PO/SC bd–tds or PRN	Parkinsonism, dementia, QT
Metoclopramide	Gastric stasis, reflux	10–20 mg PO/SC tds; up to 120 mg/day CSCI	Complete obstruction, within 3 days of GI surgery
Hyoscine butylbromide	GI/urinary colic; secretions; obstruction	10 mg PO tds; 20 mg SC, up to 60 mg/day CSCI	Glaucoma, outflow obstruction, ileus
Olanzapine	Nausea, delirium	2.5 mg → 10 mg PO at night	Diabetes, Parkinsonism
Omeprazole / pantoprazole	Dyspepsia, NSAID gastroprotection	20 mg OD / 40 mg OD	Warfarin, ketoconazole interactions
Bisacodyl	Constipation	10–20 mg PO at night; 10 mg PR	Avoid in obstruction
Lactulose	Constipation; hepatic encephalopathy	15 ml bd; 30–50 ml tds titrated	Bloating, cost
Tranexamic acid	Bleeding from small vessels	1–1.5 g PO tds–qid	Thromboembolism
Zoledronic acid	Hypercalcaemia; bone pain, SREs	4 mg in 100 ml over 30 min; repeat 3–4-weekly for SREs	Renal impairment
Metronidazole	Malodorous wound; anaerobes	Topical gel/crushed tablet OD–BD; 400 mg PO tds	Warfarin
Midazolam	Agitation, seizures, haemorrhage, hiccup	2.5–10 mg SC PRN; 10 mg SC for fits; 5–10 mg IV/IM for haemorrhage; 10–60 mg/24 h CSCI	Additive sedation

Equivalent corticosteroid doses (IAPC)	
Dexamethasone 2 mg	= prednisolone 15 mg = hydrocortisone 60 mg = methylprednisolone 12 mg
Choosing	Dexamethasone — high potency per tablet, little mineralocorticoid effect, less fluid retention; prednisolone — less proximal myopathy; hydrocortisone — marked salt and water retention
Trial	High dose for 3 days to test benefit, then taper to the lowest effective dose

Other pains (IAPC)	Treatment
Skeletal muscle spasm	Diazepam (small doses) or baclofen 5 mg tds

Colic (bowel, ureter)	Hyoscine butylbromide — colic responds poorly to morphine
Liver capsule pain	Dexamethasone 4–8 mg
Cardiac pain, oesophageal spasm	Nitrates or nifedipine

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapters 6 and 29; doses as printed in the IAPC formulary — always check current product information. Summarised and adapted in our own words for MD/DNB revision.

7.17 Opioid Conversion Calculations — the IAPC Method

Section 7 contents ›

IAPC-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- **Q** A patient on SR morphine 90 mg 12-hourly cannot swallow. Calculate the fentanyl patch dose. Show your working.
- **Q** Convert codeine 40 mg 4-hourly to oral morphine, and SR morphine 60 mg bd to subcutaneous morphine.

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Calculate equianalgesic doses and convert between opioids and routes (CD2.1.3, PD3, PD8)

All conversions start from the **total 24-hour oral morphine dose (M_{24})**, including breakthrough doses taken. Equations first, then worked calculations, using the IAPC rules.

STEP 0

Total 24-hour oral morphine

M_{24} (mg) = dose per administration × number of doses in 24 h

4-hourly = 6 doses/day; 12-hourly (SR) = 2 doses/day. Add the rescue doses actually used.

RULE 0

Starting and titrating immediate-release (IR) oral morphine

Starting dose = 5–10 mg IR every 4 hours (6 doses/day) · double dose at bedtime

New 4-hourly dose = old 4-hourly dose × 1.5 (increase by up to 50% per 24 h if pain persists)

Titration is always done with IR morphine (IAPC); SR is used only once the 24-h need is known. The calculations below work from IR or SR — only M_{24} matters.

Example — IR morphine 10 mg 4-hourly, still in pain, using 2 rescue doses of 10 mg

1. M_{24} = 10 × 6 + 2 × 10 = 80 mg
2. New 4-hourly dose = 10 × 1.5 = 15 mg (or 80 ÷ 6 ≈ 13 mg → 15 mg)

→ IR morphine 15 mg 4-hourly (30 mg at bedtime); rescue 15 mg

RULE 1

Codeine → oral morphine

Oral morphine (mg/24 h) = codeine (mg/24 h) ÷ 10

Conversion factor between codeine and morphine = 10.

Example — codeine 40 mg 4-hourly

1. Codeine/24 h = 40 × 6 = 240 mg
2. Morphine/24 h = 240 ÷ 10 = 24 mg

→ Oral morphine about 5 mg 4-hourly (24 ÷ 6 = 4, rounded to the 5 mg tablet)

RULE 2

Immediate-release (4-hourly) → sustained-release (12-hourly)

SR dose (12-hourly) = M_{24} ÷ 2

Titrate on IR first; never start with SR. IR works ~4 h, SR ~12 h.

Example — IR morphine 20 mg 4-hourly

1 M_{24} = 20 × 6 = 120 mg
2 SR dose = 120 ÷ 2 = 60 mg

→ SR morphine 60 mg bd

RULE 3

Breakthrough (rescue) dose

Breakthrough dose (IR) = M_{24} ÷ 6 (= the regular 4-hourly dose)

Give as often as needed. If rescue doses are needed more than twice a day regularly, increase the regular dose (IAPC: increase the 4-hourly dose by up to 50% per 24 h).

Example — SR morphine 60 mg bd

1 M_{24} = 60 × 2 = 120 mg
2 Breakthrough = 120 ÷ 6 = 20 mg

→ IR morphine 20 mg as needed

Example — IR morphine 10 mg 4-hourly (not on SR)

1 M_{24} = 10 × 6 = 60 mg
2 Breakthrough = 60 ÷ 6 = 10 mg

→ IR morphine 10 mg as needed — the same as the regular 4-hourly dose

RULE 4

Oral morphine → subcutaneous morphine

SC morphine (mg/24 h) = M_{24} ÷ 2 · Intermittent SC dose (4-hourly) = SC_{24} ÷ 6

Subcutaneous morphine is 2 times as potent as oral morphine. Use CSCI, or a scalp-vein needle for intermittent doses at home.

Example — SR morphine 60 mg bd, now unable to swallow

1 M_{24} = 60 × 2 = 120 mg
2 SC_{24} = 120 ÷ 2 = 60 mg
3 Intermittent SC = 60 ÷ 6 = 10 mg

→ SC morphine 60 mg/24 h by CSCI, or 10 mg SC 4-hourly

Example — IR morphine 15 mg 4-hourly, now vomiting

1 M_{24} = 15 × 6 = 90 mg
2 SC_{24} = 90 ÷ 2 = 45 mg
3 Intermittent SC = 45 ÷ 6 = 7.5 mg

→ SC morphine 45 mg/24 h by CSCI, or 7.5 mg SC 4-hourly

RULE 5

Oral morphine → transdermal fentanyl patch (Stanford steps, as in IAPC)

Fentanyl (mg/24 h) = M_{24} ÷ 100 → × 1000 = µg/24 h → ÷ 24 = patch strength (µg/h)

Combined: Patch (µg/h) = M_{24} × 10 ÷ 24 (≈ M_{24} ÷ 2.4)

Parenteral fentanyl is 100 times as potent as oral morphine. Reference point: 60 mg oral morphine/24 h (10 mg 4-hourly) ≈ 25 µg/h patch, changed every 3 days.

Example — SR morphine 90 mg bd

1 M_{24} = 90 × 2 = 180 mg
2 Fentanyl/24 h = 180 ÷ 100 = 1.8 mg = 1800 µg
3 Hourly = 1800 ÷ 24 = 75 µg/h

→ Apply a 75 µg/h patch with the last SR morphine 90 mg dose, then stop SR morphine. Keep IR morphine 30 mg (180 ÷ 6) for breakthrough pain — 2–3 doses may be needed in the first day

Example — IR morphine 10 mg 4-hourly (stable, cannot take tablets reliably)

1. M_{24} = 10 × 6 = 60 mg
2. Fentanyl/24 h = 60 ÷ 100 = 0.6 mg = 600 µg
3. Hourly = 600 ÷ 24 = 25 µg/h

→ 25 µg/h patch; continue IR morphine 4-hourly for the first 12 h (patch takes 12–24 h to work); rescue 10 mg

Rule of thumb (IAPC): M_{24} ÷ 3 → choose the nearest patch strength

180 ÷ 3 = 60 → a 50 or a 75 µg/h patch. With the lower (50) patch, expect more rescue doses. The same ÷ 3 rule is used for children (e.g. 20 mg 4-hourly → 120 ÷ 3 = 40 → 50 µg/h patch).

REVERSE

Fentanyl patch → oral morphine

M_{24} (mg) = patch (µg/h) × 24 ÷ 1000 × 100 = patch × 2.4

25 µg/h × 2.4 = 60 mg oral morphine/24 h; breakthrough = 60 ÷ 6 = 10 mg IR.

Patch (µg/h)	12	25	50	75	100
Oral morphine (mg/24 h)	30	60	120	180	240
Breakthrough IR morphine (÷ 6)	5	10	20	30	40

SAFETY

Fentanyl patch — practical points (IAPC)

- Control pain with oral morphine first; never start a patch in an opioid-naive patient or for acute or unstable pain.
- Peak levels at 12–24 h, so apply the patch with the last SR dose; a skin depot persists ~24 h after removal.
- Apply to dry, non-inflamed, non-irradiated, hairless skin; fever, external heat or a hot bath increase absorption.
- Reduce laxatives (less constipation than morphine). About 1 in 10 patients have withdrawal symptoms when switched from morphine.
- Strengths available: 12.5, 25, 50, 75, 100 µg/h, changed every 72 h. Buprenorphine patch (where available): start 35 µg/h in naive patients, maximum 140 µg/h, not more than two patches at a time.

★ CLINICAL PEARLS & EXAM FOCUS

- Order of working in every conversion: **M_{24} → apply the rule → round to an available strength → write the breakthrough dose (÷ 6)**.
- When switching because of toxicity, give a lower starting dose and review within 24–48 h (see opioid rotation, p.270).

Source: Indian Association of Palliative Care (IAPC), Textbook for the Certificate Course in Essentials of Palliative Care, 5th ed. (2017), chapter 6 (Management of pain) and chapter 18 (Paediatric palliative care). Summarised and adapted in our own words for MD/DNB revision.

7.18 Principles of Symptom Management (EEMMA), Quality of Life & Appropriate Treatment

Section 7 contents ›

Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Describe the general principles of symptom management in advanced cancer.
- Q Define quality of life. How does palliative care improve it?
- Q What is "appropriate treatment" in a dying patient?

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Apply general principles of symptom assessment and management (CD3.1.1, CD1.2.1)
- ✓ Make ethically sound decisions on limiting disease-modifying treatment (CD9.1.3 / 9.1.4)

EXAM-ORIENTED THEORY

EEMMA — the scientific approach to any symptom

Step	What it means in practice

Evaluation	Diagnose each symptom before treating. Ask: cause (cancer, treatment, debility or a concurrent disorder)? mechanism (vomiting from hypercalcaemia differs from raised ICP)? what has been tried and failed? impact on life and sleep? Ask about "hidden" symptoms — dry mouth, taste, anorexia, itch, insomnia
Explanation	Explain the mechanism in simple words before treating — it reduces fear and improves adherence; agree treatment options with the patient
Management	Correct the correctable; use non-drug as well as drug measures; give drugs **regularly, by the clock**, for persistent symptoms; keep the regimen simple; give **written** instructions (time, drug, purpose, dose); seek a colleague's advice; never say "nothing more can be done"
Monitoring	"Review! Review! Review!" — doses of opioids, laxatives and psychotropics cannot be predicted; new symptoms arise; sometimes accept partial relief to avoid intolerable side effects
Attention to detail	An inquisitive mind that keeps asking "why?"; no unwarranted assumptions; detail matters equally for non-physical care

Quality of life

- "Quality of life is what a person says it is" — subjective satisfaction across physical, psychological, social and spiritual domains.
- Good when aspirations are matched by present experience; poor when the gap is wide. Palliative care narrows the gap — by improving reality or by helping reset expectations to what is possible.
- Rehabilitation is part of palliative care: "add life to their days, not days to their life".

Partnership with the patient

- A consultation is a meeting of experts — the patient on how they feel, the team on diagnosis and options.
- Be courteous, listen, explain, be honest, discuss options, agree priorities and goals, accept refusal, never be condescending.
- Palliative care is "low tech, high touch": patient-centred, death-accepting yet life-enhancing, concerned with healing rather than curing.

EXAM-ORIENTED THEORY

Appropriate treatment

- Treatment that gives no net benefit may, ethically and legally, be withheld or withdrawn; the goal shifts to comfort. A doctor has neither the duty nor the right to prescribe a lingering death.
- The question is not "treat or not treat" but "what is the most appropriate treatment", given the patient's biological prospects, the aim and burdens of each treatment, and personal circumstances.
- Measures for acute illness (NG tubes, IV fluids, antibiotics, CPR, ventilation) carry a patient through a crisis towards recovery; in someone irreversibly close to death their burdens usually exceed benefits.
- Near death, loss of interest in food and fluid is part of letting go — do not force it.

Pain is increased by	Pain is decreased by
Discomfort, insomnia, fatigue	Relief of other symptoms, sleep
Anxiety, fear, anger, sadness, depression	Understanding, companionship, relaxation, elevated mood
Boredom, mental isolation, social abandonment	Creative activity, analgesics, anxiolytics, antidepressants

★ CLINICAL PEARLS & EXAM FOCUS

- Four cardinal ethical principles — autonomy, beneficence, non-maleficence, justice — applied against respect for life and acceptance that death is inevitable; see p.348.
- Double effect: correctly used morphine is a very safe drug; the intention must always be relief of suffering, never death.

Trauma & Critical care Medicine

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 1 (General topics) and Part 3 (General principles). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.19 Hope, Uncertainty and the Withdrawn or Difficult Patient

Section 7 contents ›
Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q How will you help a patient live with uncertainty about prognosis? Describe practical coping strategies.
- Q Discuss the causes and management of the withdrawn patient.
- Q What is meant by the "difficult patient"? Explain transference and countertransference.

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Communicate about prognosis and uncertainty; maintain realistic hope (CD9.1.6–9.1.8)
- ✓ Recognise psychological responses, defence mechanisms and psychiatric morbidity (CD7.1.1–7.1.3, CD7.2)

EXAM-ORIENTED THEORY

Hope

- "Hope is an expectation greater than zero of achieving a goal" — hope needs an object. Set realistic goals together; break an unrealistic ultimate goal ("I want to walk again") into achievable mini-goals.
- Telling a painful truth does not destroy hope — hope of cure is replaced by other hopes; near death it focuses on *being* rather than achieving, on relationships and on God or a higher being. It is always realistic to hope for a peaceful death.

Hope decreases with	Hope increases with
Feeling devalued; abandonment and isolation	Feeling valued; meaningful relationships
A "conspiracy of silence" or of words; "there's nothing more I can do"	Reminiscence, humour
No direction or goals; unrelieved pain	Realistic goals; pain and symptom relief

EXAM-ORIENTED THEORY

Coping with uncertainty — four strategies

Strategy	When	How
A rolling horizon	Expected survival ≥4–6 months	Plan confidently for the next 2–3 months; if still as well after a month, fill the diary for another month, and so on
Hope for the best, plan for the worst	About 2–3 months	A "cross-eyed" approach — one eye on plans if things go well, the other on alternatives if they do not
Reaching anniversaries	Outlook very poor but still hoping for time	Aim for special events or anniversaries one at a time
One day at a time	Last few weeks	Helpful near death; in less ill patients suggest 1–2 weeks at a time to avoid wasted opportunities

Answer "How long have I got?" honestly: nobody knows. Ask what prompted the question, acknowledge how hard uncertainty is, then discuss the strategies above. Patients "extremise" ranges — "2–3 months or 2–3 years" is remembered as one or the other. Deteriorating month by month → months; week by week → weeks; day by day → days.

Response to loss	Features	Typical duration
Disruption	Disbelief, denial, shock, numbness	Days → weeks
Dysphoria	Anxiety, insomnia, poor concentration, anger, guilt, sadness, depression	Weeks → months
Adaptation	Implications confronted, new goals, hope refocused, activities resumed	Months

The withdrawn patient — causes

- **Personality**
- **Pathological** — brain tumour, cerebrovascular disease, delirium/dementia, hypothyroidism
- **Pharmacological** — oversedation, tardive dyskinesia
- **Psychological** — anger, collusion, distrust ("no point talking"), fear ("too painful"), guilt, shame ("too embarrassed")
- **Psychiatric** — depression, paranoia ("too dangerous")

The difficult patient

- Say "I find Mrs B difficult to look after", not "Mrs B is difficult" — the problem is primarily ours.
- Reasons: the patient or relative is perceived as unpleasant, ungrateful, critical, demanding, manipulative or over-dependent; behaviour is withdrawn, angry, depressed or antagonistic; symptoms such as gross disfigurement, malodour, poor response or somatisation.
- **Transference**: the patient's behaviour evokes negative feelings in us because of our past experiences.

Approach

- Acknowledge the difficulty: "We seem to be finding it hard to talk."
- Offer an invitation they can accept or decline; follow up any clue gently but firmly
- Check frequency and intensity of low mood (psychiatric illness?); seek specialist help if stuck

Countertransference: our personality evokes negative feelings in the patient. Both sense the "vibes".

- Management: share the difficulty with the team, explore reasons, agree a written plan with short-term goals and time to be spent, accept that some problems cannot be solved.

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Depression affects about 15% of patients — ask: "What has your mood been like lately?", "Are you depressed?". Paranoid states may be due to steroids, biochemical upset, cerebral metastases or psychological factors.
- ▸ Denial is "a psychological shock-absorber"; intervene only if it blocks treatment, planning or relationships (see 7.2).

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 1 (Hope) and Part 2 (Psychosocial aspects of care). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.20 Care of the Relatives and Dying at Home

Section 7 contents ›
Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Discuss the care of the relatives of a dying patient.
- Q What is needed for a patient to die at home? How will you prepare the family?

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Support families and caregivers; plan care at home (CD7.4.1 / 7.4.2, CD8.3.1–8.3.4)

A contented family increases the likelihood of a contented patient. The doctor must start and maintain communication — families hesitate to "trouble a busy doctor".

EXAM-ORIENTED THEORY

Telling the relatives

- "Please don't tell him, doctor" is usually an initial shock reaction born of love and fear — not a reason to say nothing to the patient. Help the family move towards openness.
- Confidentiality: relatives are told with the patient's permission, not the other way round; the family cannot forbid the doctor from talking to the patient.
- Joint interviews (patient, relative, doctor, nurse) at diagnosis and later prevent collusion; the nurse can clarify later. Also see patient and family separately. Do not give the family the whole truth at once — they also need time.

Situation	Good practice
Inpatient admission	May feel like failure to the family — praise how long they have coped; encourage visiting and helping (pillows, meals, bathing); teach how to "just be there" (read, knit, watch TV together); arrange overnight stay if needed
Planning discharge	Some patients improve once symptoms are controlled; a day or weekend at home tests feasibility; prevent over-protection — support independence (driving, outings); say whom to call in an emergency
Explaining treatment	Warn that if swallowing fails, medicines will be given by injection or suppository; if pneumonia develops it will be treated for comfort, not with antibiotics
After the death	Bereavement has a morbidity and a mortality — allow relatives to voice "if only…" guilt

EXAM-ORIENTED THEORY

Dying at home — requirements

- A fit relative who can cope
- Nurses who can visit daily or more often
- An attentive doctor who visits routinely
- A team able to respond quickly to new problems
- Guaranteed rapid admission in a major crisis
- Give the date of the next visit ("next Wednesday around midday") rather than "call if there are problems" — families otherwise feel in limbo. Real 24-hour availability is rarely abused once tested.

- Prepare relatives for delirium and death rattle; plan subcutaneous (CSCI) or rectal routes; keep suppositories or injections at home for seizures or (in head and neck cancer) massive haemorrhage.
- Preferences change: in one series the wish for home care fell to about 50% and fewer than a third died at home. Living alone, an unfit carer or a prolonged terminal illness often lead to admission.

★ CLINICAL PEARLS & EXAM FOCUS

► Planning death at home: algorithm p.329; Kerala home-care model p.370; caregiver burden p.376.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 1 (Dying at home) and Part 2 (Care of the relatives). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.21 Religious Needs at the End of Life

Section 7 contents ›
Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q Discuss the religious and cultural needs of dying patients of different faiths.

Q Distinguish spirituality from religion. List the indicators of spiritual distress.

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Define spirituality and religion; assess and support spiritual and existential needs (CD7.3.1)

Religion may be **life-enhancing** (supportive) or **life-escaping** (adds fear). A religious label does not guarantee a fixed set of beliefs — listen and do not assume. Spirituality concerns meaning and purpose, connectedness and a right relationship with God or ultimate reality; religion is a shared framework of beliefs and rituals that expresses it.

Issue	Points to remember
Modesty	Many Asian patients and Muslims are distressed by intimate care from a carer of the opposite sex; many Asian women dislike undressing for examination
Medication	Some Buddhists and Hindus may decline analgesics or sedatives to keep a clear mind
Blood transfusion	Refused by Jehovah's Witnesses
Organ donation / autopsy	Some restrictions for Jews; autopsy acceptable to Jews and Muslims only if legally required, distasteful to many Hindus
Disposal of the body	Burial within 24 h for Jews and Muslims (no embalming); cremation for Sikhs and most Hindus; burial or cremation for Christians and Buddhists
After-life beliefs	Resurrection — Christians, Jews, Muslims; rebirth — Buddhists; reincarnation — Hindus and Sikhs
Diet	No alcohol for Muslims and Sikhs (check alcohol-containing syrups); many Hindus, Sikhs and some Buddhists vegetarian; most Hindus avoid beef; Sikhs avoid halal meat; Jews and Muslims avoid pork and shellfish; Orthodox Jews eat only kosher meat

Faith	Around death
Hinduism	Morning bath in running water and prayer at sunrise/sunset matter; some prefer to die on the floor near Mother Earth; family places water (often Ganga jal) on the lips; relatives usually wash the body; mourning 7–41 days with the extended family
Islam	Prayer five times a day after washing; Qur'an read and articles of faith whispered to the dying; family visits often; staff should not wash the body — use gloves; arms in prayer position, head turned to the right, body facing Mecca; washing by family or Muslim undertaker of the same sex
Christianity	Priest or chaplain; Bible readings, prayer and sacraments (reconciliation, anointing, communion); prayers of commendation after death
Sikhism	No priests; scriptures read near death and hymns over the body at home; staff may handle the body but family usually wash it; white is the colour of mourning (about 10 days)
Buddhism	May wish to stay clear-headed and decline sedating drugs; contact a monk if wished; anyone may prepare the body
Judaism	Community often supports the patient and may stay with the body; staff wear gloves; community prepares the body for burial; 3 days of intense grief, then periods of 7 and 30 days

Indicators of spiritual distress

- Hopelessness, meaninglessness ("What's the point?"); intense suffering, loneliness
- Remoteness from or anger at God ("Why me? What have I done?"); undue stoicism
- Guilt or shame (illness as punishment), being unforgiving of self or others; vivid nightmares

★ CLINICAL PEARLS & EXAM FOCUS

- Death is not the ultimate tragedy — depersonalisation is: dying in an alien place, cut off by a conspiracy of silence, in hopelessness.
- Useful questions: "What or who helps you most when life is hard?" "What part does religion play in your life?" FICA tool: p.307.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 2 (Spiritual care; Religious needs). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.22 Bereavement — Reactions, Models of Grief and Risk Assessment

Section 7 contents ›
Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Describe the models of grief. What is the dual process model?
- Q How will you identify relatives at high risk of complicated bereavement?
- Q How does the death of a parent affect children, and how can the surviving parent be helped?

◉ CBME COMPETENCIES — the postgraduate should be able to:

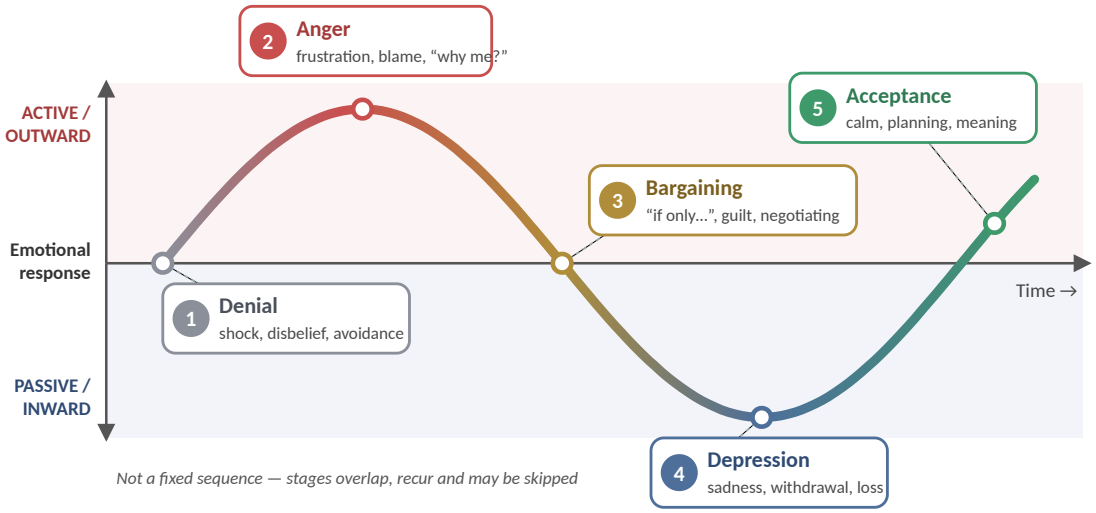
- ✓ Describe theories of grief, normal and complicated grief, and bereavement support (CD7.1.4 / 7.1.5)

Grief is not only emotional — it is behavioural, cognitive, physical, social and spiritual; a transition in which the bereaved assimilate the loss and learn to live without the deceased's physical presence. Bereavement carries real morbidity and mortality.

Domain	Common reactions
Emotional	Waves of sadness and despair, anxiety, guilt, anger (at deceased, family, carers, God), loneliness, loss of enjoyment, relief
Behavioural	Agitation and searching, fatigue, crying
Attitudes	Self-reproach, low self-esteem, hopelessness, unreality, suspicion, social withdrawal; yearning, pre-occupation, idealisation, sensing or "hallucinating" the deceased
Physical	Poor appetite and weight change, insomnia, aches, palpitations, dry mouth, sighing; more alcohol, tobacco and psychotropics; infections and stress illness

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FIGURE 7.22 — FIVE STAGES OF GRIEF (KÜBLER-ROSS): A TEACHING ADAPTATION



How to read it: the vertical axis shows how emotion is *expressed* — anger is active and turned outward (peak); depression is passive and turned inward (trough). **Exam points:** Kübler-Ross (*On Death and Dying*, 1969) described these responses in **dying patients**; her own chart showed overlapping responses, including hope, partial denial, preparatory grief and decathexis — this wave is a later teaching adaptation. Stages are **not linear, not universal and not a checklist**: people skip, repeat and move back and forth. For bereavement, the dual process and task (Worden) models are more useful (table below).

EXAM-ORIENTED THEORY

Models of grief

Model	Key idea
Traditional (phase) model	Numbness → separation and pain (pining, searching) → despair → acceptance → resolution and re-organisation. Useful themes, but grief is not an orderly sequence — avoid judging where someone "should" be
Continuing bonds	Healthy adjustment does not require severing the bond; new connections with the deceased are built, so there may be no fixed "closure"
Dual process (Stroebe & Schut)	People oscillate between **loss-oriented** coping (memories, pining) and **restoration-oriented** coping (new roles, distraction). Only-loss = chronic grief; only-restoration = absent grief
Multidimensional	Loss affects emotional, social, physical, lifestyle, practical, spiritual and identity dimensions at once; build on each person's strengths

EXAM-ORIENTED THEORY

Risk assessment — predictors of a difficult bereavement

Factor	Higher risk when
Mode of death	Untimely, unexpected or unduly disturbing (even after a terminal illness)
Relationship	Highly ambivalent (persistent guilt) or highly dependent
Perceived support	Unable to share feelings; feels isolated
Anticipatory grief	Little open talk before death; excessive denial or anger
Concurrent stresses	Finances, unemployment, dependants, no time or space to grieve
Previous losses / health	Unresolved earlier losses; physical or mental illness; alcohol or drug misuse; suicidal behaviour

The strongest protective factor is a supportive family and friends who allow grief to be expressed for as long as needed. Give written information about grief and local help.

EXAM-ORIENTED THEORY

Children bereaved of a parent

- Most affected by: adverse social and financial change, the relationship with the surviving parent, and the emotional climate; also confusion when not told, new carers, moving home or school, early remarriage.
- Responses — physical (sleep, appetite, regression to wetting or soiling, flare of asthma/eczema, mimicking the parent's symptoms), emotional (separation anxiety, clinging, mood swings, guilt), cognitive (poor concentration and memory, "what's the point"), behavioural (regression, risk-taking, a brave face).

- **Treat the parent as a colleague**: explain normal reactions; suggest books, drawing, writing and a memory box or scrapbook; expect death-themed play; be honest about one's own feelings ("I'm crying because I'm sad"); involve teachers and relatives; avoid "ought" and "should"; parents must look after themselves too.

★ CLINICAL PEARLS & EXAM FOCUS

- Children's grief by age group: see 7.9; bereavement algorithm p.306; grief theories essay p.353.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 2 (Bereavement; Children and bereavement). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.23 Requests for Euthanasia — Responding in Practice

Section 7 contents ›
Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

Q A patient asks you to "end it all". How will you respond?

Q What is not euthanasia? List the pragmatic arguments against legalising euthanasia.

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ Discuss futility, euthanasia and ethical decision-making at the end of life (CD8.3.1–8.3.4, CD9.1.3 / 9.1.4)

DEFINITION **Euthanasia**: a deliberate intervention undertaken with the express intention of ending a life to relieve intractable suffering — voluntary if at the person's request or with consent, otherwise non-voluntary. Indian law is covered on p.349.

Euthanasia is NOT

- Allowing nature to take its course
- Stopping biologically futile treatment
- Stopping treatment whose burdens outweigh benefits
- Using morphine or other drugs to relieve pain
- Sedation for intractable distress in a dying patient

Avoid the terms "passive" and "indirect" euthanasia — withdrawing futile treatment or giving analgesia is not deliberate acceleration of death.

Why patients ask

- Unrelieved pain, breathlessness or vomiting — or fear of them (suffocation, choking)
- Fear of slow deterioration or of being kept alive on machines
- Temporary despair after bad news (adjustment disorder)
- Demoralisation; depressive illness
- Feeling a burden, unwanted or no longer in control
- A fixed hopelessness rooted in a view of life with no meaning beyond death

Persistent requests are uncommon, and most who ask for help to die are asking for help to live — hear the "cry for life" and address each reason. Except for the last two reasons, the situation can usually be improved enough for the person to change their mind.

Pragmatic reason against euthanasia	Comment
Prognosis is often uncertain	Some patients live years longer than expected
Patients often change their minds	Despair is frequently transient
Many requests stem from poor symptom relief	Requests stop when symptoms are relieved
Feeling useless or a burden	Good palliative care restores direction and connectedness
Persistent requests often mean depression	Depression needs specific treatment
The "slippery slope"	Voluntary may extend to non-voluntary and to non-terminal patients
Pressure on the vulnerable	Elderly and dying may feel at risk; families may press; budgets may tempt
Less incentive to improve care	The option removes pressure to raise standards

★ CLINICAL PEARLS & EXAM FOCUS

- Palliative care and euthanasia are mutually exclusive philosophies; palliative care neither hastens nor postpones death.
- Algorithm: approach to a request for hastened death p.282.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 1 (Ethical considerations; Euthanasia). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.24 Dyspepsia, Gastric Stasis, Cough and Hiccup

Section 7 contents ›
Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Classify dyspepsia in advanced cancer and outline management of each type.
- Q Gastric stasis — clinical features and management. Why should prokinetics and antimuscarinics not be combined?
- Q Protussives versus antitussives. Outline the management of intractable hiccup.

CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Manage GI symptoms in advanced illness (CD3.2)
- ✓ Manage cough and hiccup (CD3.3.2, CD3.5.1)

EXAM-ORIENTED THEORY

Dyspepsia — four therapeutic types

Type	Typical cause	Treatment
Small stomach capacity	Large gastric cancer, massive ascites, hepatomegaly ("squashed stomach")	"Small and often" — 5–6 small meals; separate fluids from solids; antiflatulent after meals
Gassy	Aerophagia, anxiety	Antiflatulent (dimeticone, e.g. with an antacid)
Acid	Peptic ulcer, reflux, NSAIDs, steroids	Antacid, H2 blocker or PPI (PPI for NSAID gastritis)
Dysmotility	Functional, opioids, antimuscarinics	Metoclopramide — acid suppression does not help

EXAM-ORIENTED THEORY

Gastric stasis

- Main cause of about 25% of nausea and vomiting in advanced cancer. Features: early satiety, post-prandial fullness, bloating, belching, hiccup, heartburn, nausea and large-volume vomits; succussion splash (needs >400–500 ml fluid plus gas).
- Causes: functional dysmotility, constipation, drugs (opioids, antimuscarinics, aluminium hydroxide, levodopa), cancer of the pancreatic head, paraneoplastic or diabetic autonomic neuropathy, retroperitoneal disease, cord compression.
- Treat with explanation and a prokinetic — **metoclopramide** (domperidone in parkinsonism). **Do not combine prokinetics with antimuscarinics**: antimuscarinics block the cholinergic final pathway through which prokinetics act.

Cough

- Causes: smoking, asthma, COPD, infection, reflux, heart failure, intrathoracic cancer, ACE inhibitors.
- Correct the correctable (bronchodilators, antibiotics); teach effective coughing (hard lying flat), physiotherapy, steam inhalations.
- **Wet cough, able to cough → protussives** to aid expectoration: nebulised 0.9% saline, steam/benzoin inhalations, mucolytics (carbocisteine, acetylcysteine).
- **Dry cough, or too weak to cough → antitussives**: simple linctus, opioids (pholcodine, codeine, morphine), nebulised bupivacaine (rarely).

Hiccup

- Causes: gastric distension (commonest), reflux, diaphragmatic or phrenic irritation, uraemia, infection, CNS tumour.
- **Acute**: pharyngeal stimulation (cold drink, sugar, nebulised saline); reduce gastric distension (peppermint water, dimeticone, metoclopramide 10 mg); raise pCO_2 (rebreathing, breath-holding); baclofen 5 mg, nifedipine 10 mg, midazolam 2 mg IV; central suppression — haloperidol 5–10 mg, chlorpromazine 10–25 mg, sodium valproate.
- **Maintenance**: distension — dimeticone or metoclopramide 10 mg qds; reflux — prokinetic ± H2 blocker/PPI; other — baclofen 5–20 mg tds, nifedipine 10–20 mg tds, haloperidol 1.5–3 mg at night, valproate 200–1000 mg at night, midazolam 10–60 mg/24 h CSCI as a last resort.

Antisecretory for death rattle	Stat SC dose	CSCI per 24 h
Hyoscine hydrobromide	0.4–0.6 mg	1.2–2.4 mg
Hyoscine butylbromide	20 mg	20–40 mg

Glycopyrronium	0.2–0.4 mg	0.6–1.2 mg

★ CLINICAL PEARLS & EXAM FOCUS

- Acute tracheal compression or massive haemorrhage into the trachea: IV midazolam or diazepam 5–20 mg until unconscious (IM midazolam or rectal diazepam if no IV), and stay with the patient — see p.288.
- Hiccup algorithm p.324; death rattle p.310.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 4 (Alimentary and respiratory symptoms). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.25 Urinary Symptoms in Advanced Illness

Section 7 contents ›

Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Describe the autonomic innervation of the bladder and the effect of drugs on bladder function.
- Q Outline the causes and management of urgency, retention and bladder spasm in advanced cancer.

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Manage urinary retention, incontinence and bladder symptoms (CD5.1.5 / 5.1.6, CD10.1)

Term	Definition
Frequency	Passing urine ≥7 times by day and ≥2 times at night
Urgency / urge incontinence	Strong sudden desire to void / involuntary loss with that desire
Detrusor instability	Uninhibited detrusor contraction → frequency by day and night, urgency, urge incontinence
Stress incontinence	Leakage on coughing, sneezing, laughing, lifting (urethral sphincter incompetence — multiparity, menopause, hysterectomy)
Dysuria / hesitancy	Pain during or after voiding (urethral burning or bladder spasm) / long delay before voiding starts

Innervation

Supply	Sphincter	Detrusor
Sympathetic (T10–L1), noradrenaline	Contracts	Relaxes
Parasympathetic (S2–4), acetylcholine	Relaxes	Contracts

"You pee with your parasympathetics; you stop with your sympathetics." The voluntary urethral sphincter is supplied by the pudendal nerve (S2–4).

Drug effects

- **Antimuscarinics** relax the detrusor and tighten the bladder neck → hesitancy, retention
- **Opioids** reduce bladder sensation and raise sphincter and detrusor tone — occasionally hesitancy or retention (especially spinal analgesia)
- **Prostaglandins** increase and **NSAIDs** decrease detrusor sensitivity
- Topical/oral oestrogen may help post-menopausal urge symptoms (not stress incontinence)

Symptom	Causes to consider	Management
Urgency, frequency, incontinence	Cystitis, tumour or radiation cystitis, hypercalcaemia or diabetes (polyuria), sacral plexopathy, cord or brain disease, diuretics, cyclophosphamide	Treat infection; "bladder drill" (void 2-hourly by the clock); oxybutynin 2.5–5 mg bd–qds, tolterodine 1–2 mg bd, or amitriptyline/imipramine 25–50 mg at night
Hesitancy, retention	Prostate enlargement, bladder-neck infiltration, presacral plexopathy, cord compression, loaded rectum, weakness, antimuscarinics, opioids, spinal analgesia	Exclude faecal impaction; suprapubic pressure, running tap, warm bottle on the abdomen; α-blocker (e.g. indoramin) or bethanechol; catheterise if needed
Bladder spasm	Infection, tumour, radiation fibrosis, catheter balloon irritation or sludging, anxiety	Change catheter or reduce balloon volume; treat infection (antibiotic, washouts); antimuscarinic; flavoxate 200 mg tds–qds if antimuscarinics not tolerated

Discoloured urine	Possible causes
Orange / yellow	Jaundice, nitrofurantoin
Red / pink	Haematuria — but also beetroot, rhubarb, rifampicin, doxorubicin, dantron-containing laxatives, phenolphthalein in alkaline urine
Blue	Methylene blue; Pseudomonas (pyocyanin) in alkaline urine
Dark	Metronidazole

★ CLINICAL PEARLS & EXAM FOCUS

- In a delirious, debilitated patient with new urinary symptoms, always do a rectal examination — faecal impaction is a common, reversible cause.
- Catheter care: 7.11; MS bladder management: 7.4.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 4 (Urinary symptoms). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.26 Drug Profiles — Antacids, Corticosteroids, Benzodiazepines, Haloperidol, Antimuscarinics

Section 7 contents ›
Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q Indications for corticosteroids in advanced cancer. When can steroids be stopped abruptly?
- Q Compare hyoscine butylbromide and hyoscine hydrobromide.
- Q Uses of midazolam in palliative care.

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Prescribe corticosteroids, benzodiazepines, antipsychotics and antimuscarinics rationally (CD2.3.3, CD2.5.1)

PROFILE

Antacids

- Magnesium salts loosen and aluminium salts constipate — most products mix both. Some (Gaviscon liquid, magnesium trisilicate) are high in sodium — caution in heart failure. Regular sodium bicarbonate → sodium load and alkalosis; calcium carbonate → hypercalcaemia.
- Alginate forms a raft against reflux (works less well with acid suppressants or antifoams); dimeticone is an antifoam that eases bloating and belching. Give 1 h after meals; separate from enteric-coated tablets.

PROFILE

Corticosteroids

Use	Examples (dexamethasone doses)
Specific	Raised ICP 8–16 mg; spinal cord compression 16–32 mg; nerve compression; SVC obstruction; lymphangitis; stridor; obstruction of bronchus, ureter or bowel; radiation inflammation
Pain relief	Nerve compression, bone pain, raised ICP headache
General	Appetite and well-being 2–4 mg (prednisolone 15–30 mg); anti-emetic

- Dexamethasone is ~7× as potent as prednisolone (2 mg ≈ 15 mg), acts 36–54 h — give once daily in the morning. Watch for moon face, myopathy, hyperglycaemia, oedema, insomnia, agitation.
- **Stopping**: if no benefit after 7–10 days, stop. Abrupt stop is fine after <3 weeks' treatment if relapse is unlikely. **Taper** if >3 weeks, >40 mg prednisolone (≈ dexamethasone 4–6 mg) daily, evening doses, recent long-term use or other adrenal suppression — reduce quickly to 7.5 mg prednisolone equivalent, then 1–2 mg a week. After whole-brain RT keep dexamethasone 4 mg for ≥1 week, then cut 1 mg a week. In the dying who cannot swallow, stopping abruptly is acceptable.

PROFILE

Benzodiazepines

- Delirium is treated with an antipsychotic — benzodiazepines may worsen it (exception: alcohol withdrawal).
- Night sedation: temazepam 10–40 mg; zopiclone/zolpidem; anxiety: lorazepam 1–2 mg or oxazepam 10–15 mg bd–tds; lorazepam SL for panic attacks.
- **Diazepam** (t½ 20–100 h): anxiety 2–10 mg, spasm 2–10 mg, myoclonus 5 mg — once at night; seizures 10 mg PR/IV; rectal diazepam useful in a crisis.
- **Midazolam** (t½ ~2.5 h, water-soluble, mixes in CSCI): 3× diazepam for sedation, 2× as anti-epileptic. SC: spasm/myoclonus 5 mg stat, 10–30 mg/24 h; terminal agitation 5–10 mg stat, 30–60 mg/24 h; seizures 10 mg stat, 30–60 mg/24 h. If not settled on 30 mg/24 h, add haloperidol before increasing further. Accumulates in renal failure.

PROFILE

Haloperidol and antimuscarinics

- **Haloperidol** (t½ 13–35 h, once at bedtime): anti-emetic 1.5–5 mg; antipsychotic/delirium 1.5–10 mg; anxiolytic of choice in hallucinating, paranoid or agitated delirium; less sedation and fewer cardiovascular and antimuscarinic effects than chlorpromazine, more extrapyramidal effects (akathisia). Carbamazepine halves levels.
- **Antimuscarinic effects** — "dry as a bone, blind as a bat, red as a beet, hot as a hare, mad as a hatter": dry mouth, blurred vision, heartburn, constipation, retention, palpitations. Avoid two antimuscarinic drugs together, with prokinetics, in reflux, paralytic ileus and glaucoma.
- **Hyoscine butylbromide** (quaternary): no CNS effects, no drowsiness, no central anti-emetic action, poorly absorbed orally (oral use only for colic); SC 20 mg stat; 60–120 mg/24 h for obstruction with colic (max 300 mg); 20–40 mg/24 h for death rattle. **Hyoscine hydrobromide**: crosses the BBB, sedates, anti-emetic; repeated doses may cause agitated delirium. Both act briefly — best by CSCI.

★ CLINICAL PEARLS & EXAM FOCUS

- Steroid equivalence and the IAPC formulary: 7.16; corticosteroid algorithm p.328.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 5 (Drug profiles). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

7.27 As Death Approaches — and Care for the Carers

Section 7 contents ›

Twycross-based

EXAM-STYLE QUESTIONS — MD/DNB Palliative Medicine

- Q What are the signs that a patient with advanced cancer has only days to live? What will you do?
- Q Stresses on professional carers in palliative care and strategies to prevent burnout.

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ Recognise the dying process and simplify care (CD8.3.1–8.3.4)
- ✓ Practise self-care; recognise burnout and compassion fatigue (CD7.4.1 / 7.4.2)

EXAM-ORIENTED THEORY

Signs of the last few days (unless a reversible cause)

- Profoundly weak and bed-bound
- Semi-comatose
- Unable to take tablets or swallowing with great difficulty
- Taking no more than sips of water

What to do	How
Keep visiting	Presence matters even with nothing new to offer: "The important thing now is to keep you as comfortable as possible."
Explain weakness	To the patient: "When the body is short of energy you need more rest." Gently: "A few quiet days in bed are called for." To the family: "The illness is beginning to win."
Simplify medicines	Stop non-essential drugs; give essential ones SL, PR or SC — preferably by CSCI
Keep the family informed	"He is very weak now but may still live several days"; "He could deteriorate quickly"
Control agitation	Even if this causes sedation; listen to the nurses

Palliative care arose against the attitude that "there is nothing more we can do". There is always something to do — and when skills run out, the carer's presence remains: "You matter because you are you… we will help you not only to die peacefully, but to live until you die." The dying know we are not God; they ask only that we do not desert them.

Stresses on professional carers

- Breaking bad news; failure of cure
- Repeated deaths of people we know
- Absorbing anger and grief; emotional conflicts
- Role-blurring in teams; personal idealism
- Challenges to personal beliefs

Preventing burnout

- Teamwork — shared decisions, mutual support, good communication
- Good resources and realistic goals
- Accept support, including from patients
- Adequate time off, food, rest, recreation, hobbies and spiritual refreshment
- Outside counselling or supervision when needed

★ CLINICAL PEARLS & EXAM FOCUS

- Personal sustenance needs: feeling loved, self-esteem, creativity, flexibility, humour, compassion, and the ability to accept and correct mistakes.
- Compassion fatigue: p.377; caregiver burden: p.376.

Source: Robert Twycross, Introducing Palliative Care, 4th ed. (Radcliffe Medical Press, 2003), Part 6 (Final thoughts). Summarised and adapted in our own words for MD/DNB revision; doses reflect UK practice in that edition — check current Indian formularies.

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